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Table of Contents.

THE MORTALITY FROM VIOLENCE IN AUSTRALIA.

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VIOLENCE has always been the cause of an important proportion of deaths in Australia and so merits discussion in the consideration of Australian mortality, although it may not appear at first sight a medical topic. Moreover, further large reductions in mortality rates at the younger ages can be expected to take place only if the violent and accidental causes are controlled.

The Definition of Violent Death.

Dublin and Lotka (1937) have always paid a great deal of attention to violent causes in their reviews of the mortality experience of the policy-holders of the Metropolitan Life Insurance Company of New York. They point out that the one unifying feature of many of these deaths, which include suicides, homicides, accidental poisonings and external violence, is a lowered appreciation of the sanctity of human life. Therefore, ultimately the prevention of a proportion of these deaths is a moral problem. The State has always regarded this group of deaths as important, for it has appeared to be one in which administrative action and legislation might be effective in prevention. Therefore, in any list of deaths, care must be taken to see that violence, if it is the initiating cause, is not overlooked. Acting on these principles, the designers of the International List of Causes of Death have always given a high priority to the causes in this class. Since this has been so over the whole of the period studied, it may be said that there is strict comparability between different years.

In a comparison of Australian statistics with overseas statistics, there are some special features to be noted. In time of war, deaths from enemy action have never been included in the Australian statistics. In the first World War this made little difference, since enemy action around our shores was trivial. In the second World War, however, deaths from enemy action did occur. These were not included in the official statistics, nor were any deaths of service personnel from violence or any other cause. This results in the deaths from violence in the military age groups being rather fewer than they would have been, since violence is a cause that affects the fit, and a number of fit had been withdrawn from the population as far as their records of death were concerned; but service personnel were still numbered among the numbers at risk. The death rates from violence in the years 1941 to 1945 may be regarded as spuriously low for the military ages, although the rates for 1911 to 1920 may be regarded as satisfactory. The policy regarding the armed forces has been already outlined (Lancaster and Willcocks, 1950).

Some causes of death in this class have always been few in Australia, certainly over the years of this century. Among such rare causes are deaths from judicial execution, deaths from civil commotion and deaths from the deprivation of food or drink. There is then left for study a rather mixed group of causes of death consisting of suicide, homicide and external causes such as accident and poisons. Deaths from acute poisonings are to be included in this

class of the International List, but not deaths resulting from certain chronically acting poisons, alcoholism, plumbism and some others, which are provided for elsewhere in the list. In Australia, deaths from abortion, whether criminal or not, have been referred to the class of puerperal deaths and not to this class.

There is much to be done in preventing these deaths. Although the moral aspects are quite obvious in case of homicide or suicide, they are not so well appreciated in the case of accidents. But it is evident that many road accidents are the result of a conclusion on the part of pedestrian, motorist or road engineer that it is not worth

TABLE I.

Mortality from Violence in Australia: Deaths from External Causes (Violence) in Australia by Age and Sex for Certain Periods.

Periods.	Sex.	Death from Violence at Ages (Years).										
		0 to 4.	5 to 14.	15 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	75 and Over.	Unspecified.	Totals.
1908 to 1910 ..	M.	775	566	1,104	1,070	1,202	1,232	783	510	316	50	7,608
1911 to 1920 ..	M.	2,130	2,154	3,645	4,391	4,204	4,414	3,285	1,860	1,085	204	27,372
1921 to 1930 ..	M.	2,111	2,445	4,508	4,678	4,786	4,395	3,914	2,413	1,300	151	30,701
1931 to 1940 ..	M.	1,882	2,446	5,776	4,875	4,572	4,912	4,049	3,030	2,204	54	33,800
1941 to 1945 ..	M.	983	1,132	1,814	1,502	1,802	2,079	2,145	1,544	1,442	6	14,449
1908 to 1910 ..	F.	609	290	261	196	194	172	126	179	196	1	2,224
1911 to 1920 ..	F.	1,683	829	798	841	738	677	518	484	798	24	7,390
1921 to 1930 ..	F.	1,489	989	849	880	847	814	749	693	1,003	4	8,317
1931 to 1940 ..	F.	1,330	841	1,055	952	953	1,082	900	1,204	2,135	3	10,455
1941 to 1945 ..	F.	612	365	411	432	378	516	549	684	1,615	2	5,564

TABLE II.

Mortality from Violence in Australia: The Relative Importance of Violence as a Cause of Mortality in Australia.

Period.	Sex.	The Ratio of Violent Deaths to All Deaths Given as a Percentage at Ages (Years).					
		0 to 4.	5 to 14.	15 to 44.	45 to 74.	Over 75.	All Ages.
1908 to 1910	M.	4·15	22·53	22·07	8·80	2·44	9·72
1911 to 1920	M.	3·16	21·79	20·52	8·10	2·19	8·97
1921 to 1930	M.	3·79	25·77	25·99	7·42	2·40	9·65
1931 to 1940	M.	5·31	28·78	31·16	6·74	2·67	9·56
1941 to 1945	M.	5·30	32·62	27·73	5·66	2·61	7·30
1908 to 1910	F.	4·08	13·36	4·87	2·66	2·07	3·84
1911 to 1920	F.	3·15	9·72	4·82	2·30	1·92	3·27
1921 to 1930	F.	3·45	13·42	5·46	2·37	1·97	3·41
1931 to 1940	F.	4·86	14·10	7·11	2·56	2·75	3·77
1941 to 1945	F.	4·29	15·73	6·92	2·39	2·92	3·42

TABLE III.

Death Rates per Million from Violence in Australia for Certain Periods.

Period.	Sex.	Years of Life Experienced at Ages (Years).										
		0 to 4.	5 to 14.	15 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	Over 75.	Unspecified.	All Ages.
1908 to 1910 ..	M.	1,000	446	809	996	1,411	1,757	2,341	2,593	3,487	—	1,141
1911 to 1920 ..	M.	748	429	793	1,069	1,276	1,697	2,039	2,364	3,361	—	1,087
1921 to 1930 ..	M.	670	402	844	984	1,185	1,436	1,711	1,989	3,238	—	1,006
1931 to 1940 ..	M.	661	394	937	889	978	1,231	1,532	1,851	3,534	—	986
1941 to 1945 ..	M.	600	400	(576)	(509)	(707)	956	1,318	1,753	3,658	—	794
1908 to 1910 ..	F.	803	230	199	196	255	310	455	1,002	2,477	—	360
1911 to 1920 ..	F.	602	166	174	207	239	301	374	674	2,486	—	305
1921 to 1930 ..	F.	488	167	165	186	210	285	360	630	2,311	—	283
1931 to 1940 ..	F.	486	140	177	184	204	280	346	728	3,103	—	313
1941 to 1945 ..	F.	389	134	134	149	155	232	335	710	3,409	—	309

TABLE IV.

Mortality from Violence in Australia: The Masculinity of the Mortality from Violence by Age.

Period.	The Masculinity of the Death Rates from Violence at Ages (Years).								
	0 to 4.	5 to 14.	15 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	75 and Upwards.
1908 to 1910	125	194	407	508	553	567	515	259	141
1911 to 1920	124	258	456	516	534	564	545	351	135
1921 to 1930	137	241	511	529	564	504	475	316	140
1931 to 1940	136	281	529	483	479	440	443	254	114
1941 to 1945	154	299	430[1]	342[1]	456[1]	412	393	247	107

[1] Affected by war-time statistical practices.

while to take proper care or reasonable precautions against accidents. Legislative action has done something to lessen the toll on the road, and better engineering has helped; but ultimately control is a matter of conscience. In some countries there is considerable moral stigma associated with driving a vehicle while under the influence of alcohol, in other countries there seems to be much less. In sport, some accidents may be regarded as only to be expected

TABLE V.

Mortality from Violence in Australia: The Trend of the Mortality from Violence in Australia as Measured by the Crude and Standardized Death Rates.

Period.	Sex.	Death Rates per Million per Annum.			
		Crude Death Rate.	As Standardized onto the Enumerated Population of England and Wales, 1901.	As Standardized onto the Life Table Populations Derived from the Australian Census of 1933.	As an Equivalent Average Death Rate.
1908 to 1910	M.	1,141	1,114	1,418	1,202
1911 to 1920	M.	1,087	1,042	1,322	1,181
1921 to 1930	M.	1,006	957	1,192	1,061
1931 to 1940	M.	986	903	1,117	968
1941 to 1945	M.	794	694	910	733
1908 to 1910	F.	360	368	498	315
1911 to 1920	F.	305	310	432	271
1921 to 1930	F.	283	283	398	249
1931 to 1940	F.	313	292	447	242
1941 to 1945	F.	309	259	431	205

as an inevitable risk; but it has been a surprise to find how little moral reaction has been caused when first-class professional boxers have passed into the "punch-drunk" state. (It may be noted here that post-traumatic Parkinsonism will not be entered into the class of violent and accidental deaths of the International List.) Evidence of Jokl (1941) and others tends to be minimized, and, for example, it has not been thought worth while for anyone to follow up the later careers of retired boxers to estimate the total morbidity and mortality traceable ultimately to the boxing. Now that in New South Wales boxing is regarded as an industry and compulsory insurance against death is enforced by law, it seems only a matter of time before the chronic aspects of boxing injuries are investigated. In industry, more has been done to lessen accidents following the humanitarian movements of the last century and workers' compensation and other acts in this century. Better lighting, the shielding of dangerous machinery, the protection of the worker against noxious substances, have all no doubt assisted to keep the deaths below a level of what might have been.

Several other diseases, whose ultimate causation really lies in this group of violent deaths, have been excluded from the class of the International List. A rather common disease so excluded is pneumonokoniosis, which is usually assigned to the rubric "pulmonary tuberculosis with mention of occupational disease". Tetanus supervening on a minor accident is a less common loss from the deaths of this class. The rule here has been that if the tetanus complicates a severe compound fracture or other major injury endangering life, then the tetanus is regarded as secondary and the death is coded to the accident. But if tetanus follows a trivial scratch, wound or abrasion, the death is to be assigned to the tetanus.

Violence as an Important Cause of Death.

Owing to the importance of the deaths in this class, they have been detailed by age and sex for the five periods in Table I. A great proportion of the deaths have occurred in the active years of life in the male. A vast field for preventive measures exists.

The importance of violence can be stressed in another way by showing the proportion of deaths in different age groups that are due to violence. Thus in Table II, of boys aged five to fourteen years, and also of men aged fifteen to forty-four years, over one-quarter of all deaths are due to violent causes. The proportion of deaths in each age group is usually greater in the later periods, which means that all other causes have been more effectively controlled than violence. The only exceptions to this rule are at ages forty-five to seventy-four years for males, at which the proportion of deaths due to violence has fallen.

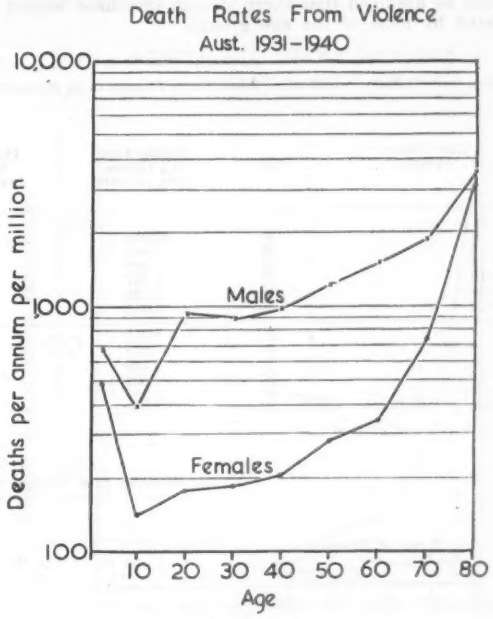

FIGURE I.

The death rates from violence in Australia for the years 1931 to 1940.

The Trend of Mortality from Violence in Australia.

In Table III, the mortality has been measured by the death rates by age and sex. They are probably strictly comparable for the different periods with the exception of those for the males of military age for the years 1941 to 1945, which are, as has already been noted for the deaths, spuriously low. These rates have been distinguished in Table III by inclusion in parentheses. War-time practices of the statistical offices possibly did not materially alter the rates of death from violence in Australia of men of military age in the first World War.

When any period is selected for study—for example, 1931 to 1940, given graphically in Figure I—it is noted that the death rates are high in the first few years of life. They then fall to a minimum for either sex in the age group five to fourteen years. For females, the rates then increase throughout life. For the males, there is a less pronounced minimum at the school ages than for the females. There is then quite a rapid rise, till at the ages fifteen to twenty-four years there is approximately one death per 1000 *per annum*. A slight fall follows, so that there is a second minimum for the male rates at ages twenty-five to thirty-four years, and this fall is followed by a continuous rise in the rates throughout the rest of the life span. This peculiarity of the death rates from violence in the twenties for males is sufficient to cause a mode in the death rates from all causes, a point that will be discussed in a later section. Of course, the fall in the rates in the later twenties is to be associated with an increased sense of responsibility with aging, and with lessened interest in motor-cycles and in dangerous sports. The trends of the death rates for any age group can also be studied in Table III. It is evident that there has been a pronounced drop in the rates for females aged under

five years, but a much smaller fall at the same ages for males. At ages above seventy-five years there has been no fall. At all other ages the decline in mortality must be regarded as disappointing, for the treatment of accident victims had greatly improved over the years of this survey. For example, the treatment of burns and compound fractures had been increasingly efficient, owing to improvements in antisepsis and asepsis, and in the most recent period to the introduction of the chemotherapeutic agents. It must be assumed that there was an increased hazard to be faced by most of the age groups.

The Masculinity of the Death Rates from Violence.

Violence would be expected from common observation or a study of occupational hazards to be a group of diseases with a high masculinity, and this is found to be so. Some individual forms of violence may tend to affect the females more than the males. Such is the case with scaldings in children aged over five years (Clements, 1952); but the male rates are usually much higher than the female rates at all ages except in old age, when they are more nearly equal. The masculinity in Table IV is merely the male rate expressed as a percentage of the female rate—that is,

TABLE VI.

Mortality from Violence in Australia: The Effect of Violence in the Production of the Mode at Ages 20 to 24 Years, in the Deaths and Death Rates, for the Years 1946 to 1948.

Age Group. (Years.)	Sex.	Deaths from All Causes, 1946 to 1948.	Deaths from Violence, 1946 to 1948.	Death Rates from Violence.	Total Death Rate from All Causes.	Death Rates from All Causes Other than Violence.
0 to 4	M.	10,616	705	605	9,113	8,508
5 to 9	M.	848	304	330	919	589
10 to 14	M.	627	271	332	769	437
15 to 19	M.	1,257	680	762	1,408	646
20 to 24	M.	1,595	919	990	1,718	728
25 to 29	M.	1,515	699	783	1,697	914
0 to 4	F.	7,969	442	396	7,139	6,743
5 to 9	F.	572	138	156	644	488
10 to 14	F.	408	74	94	517	423
15 to 19	F.	602	118	136	694	558
20 to 24	F.	999	142	154	1,080	926
25 to 29	F.	1,356	130	144	1,502	1,358

TABLE VII.

The Mortality from Violence in Australia for the Years 1931 to 1940.

Types of Violence.	Sex.	Deaths per Million per Annum at Ages (Years).								
		15 to 19.	20 to 24.	25 to 34.	35 to 44.	45 to 54.	55 to 64.	65 to 74.	75 and Over.	All Ages.
Suicide (163 to 164)	M.	48	117	174	245	357	432	384	436	178
	F.	26	49	61	82	96	88	67	42	50
Homicide (165 to 168)	M.	11	17	22	25	24	26	18	13	19
	F.	11	10	11	9	11	6	7	4	10
Venomous animals (176)	M.	3	1	2	1	3	4	3	2	3
	F.	—	—	—	—	—	—	—	—	—
Poisonings, due to food, gases and others (177 to 179)	M.	3	7	9	11	19	18	34	35	15
	F.	2	5	5	5	8	11	18	31	8
Conflagrations and accidental burns (180 to 181)	M.	14	16	17	15	21	33	77	281	37
	F.	11	8	11	12	16	18	52	138	28
Suffocation (182)	M.	2	2	3	2	4	4	7	2	8
	F.	0	0	0	1	1	2	1	0	5
Drowning (183)	M.	113	95	57	55	56	72	86	119	82
	F.	16	13	9	8	7	8	10	17	16
Firearms (184) and piercing (185)	M.	63	43	24	16	22	24	13	10	25
	F.	3	2	3	1	1	3	0	6	3
Mines and quarries (186*a*)	M.	38	28	41	42	45	46	23	6	26
	F.	—	—	—	—	—	—	—	—	—
Machinery (186*b*)	M.	17	15	20	22	23	26	12	8	15
	F.	1	0	0	0	1	0	0	0	1
Railways (186*c*)	M.	30	30	30	47	65	76	66	91	37
	F.	5	5	4	4	5	6	8	26	5
Tramways (186*d*)	M.	10	8	7	11	12	21	44	87	12
	F.	1	1	1	2	5	9	25	41	4
Automobiles (186*e*)	M.	292	465	265	239	260	327	411	611	259
	F.	54	67	48	43	69	102	175	257	67
Other land vehicles (186*f*)	M.	63	55	39	33	45	66	103	109	44
	F.	9	9	4	3	6	11	10	26	7
Water vehicles (186*g*)	M.	2	5	4	11	13	17	6	5	6
	F.	0	1	1	0	0	0	0	0	1
Air vehicles (186*h*)	M.	3	22	16	9	3	1	1	0	7
	F.	2	1	1	1	1	1	1	0	1
Other falls *et cetera* (unspecified) (186*i*)	M.	42	39	38	51	77	109	255	1,198	77
	F.	5	5	4	7	14	42	268	2,218	69
Other crushings (186*j*)	M.	20	24	32	32	42	39	28	22	25
	F.	1	1	1	0	1	0	1	1	1
Animals (188)	M.	9	6	6	5	7	10	20	22	8
	F.	0	0	0	0	1	0	1	3	1
Thirst (189), cold (190), heat (191), lightning (192)	M.	6	4	9	13	22	38	78	248	21
	F.	1	2	1	1	3	8	36	165	10
Electricity (193)	M.	10	13	16	14	11	6	4	0	10
	F.	1	1	1	2	1	0	2	1	1
Other and unstated (194, 195)	M.	41	48	55	71	94	127	178	228	70
	F.	81	9	14	21	33	30	47	122	23
War wounds (196)	M.	0	0	1	5	7	5	1	2	0
	F.	—	—	—	—	—	—	—	—	—
All forms of violence	M.	812	1,066	889	978	1,231	1,532	1,851	3,534	986
	F.	161	193	184	204	280	346	728	3,103	313

the masculinity is 100 times the male rate divided by the corresponding female rate. The masculinity tends to fall again in old age, firstly because the two sexes tend to become less active, and secondly because of the high female mortality due to falls, many of these deaths being due to complications following fracture of the femur.

The Contribution of Violence to the Fall in Mortality.

It is necessary to examine how far violence has contributed to the fall in mortality that has occurred over the period of study. In Table V are given the crude death rates from violence, and the rates standardized onto three different populations. This table may be compared with Table IX of a previous paper on mortality from all causes (Lancaster, 1951). By comparing the declines in mortality due to violence with the corresponding declines in the death rates from all causes, it may be ascertained that as a rule about 10% of the decline has been due to violence. Reduction in the death rates from violence has thus been an important factor in the decline, even though the contribution from this class has been much less than might have been expected with the increasing efficacy of modern forms of treatment of accidents. Further reductions in mortality from violence must be brought about by changes in incidence of accidents.

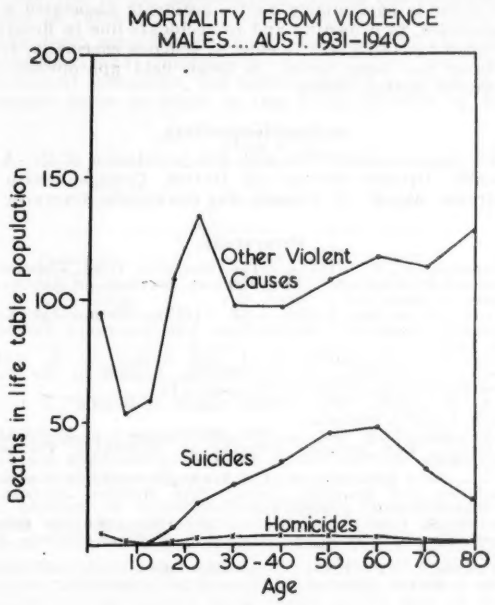

FIGURE IIA.
The deaths from violence in the life table populations, Australia, 1933 (males).

Violence as a Cause of the Mode in the Death Rates in the Early Twenties.

It has usually been found that the total death rates fall to a minimum and then increase throughout life. However, in the Australian experience there has been an increasing tendency for the rates to have a maximum in the neighbourhood of twenty years of age and then to decline to a second minimum in the late twenties. This tendency has become quite pronounced in the rates for the years 1946 to 1948, from which the most recent Australian life tables were constructed. This mode has been a topic for discussion for some time (Wickens, 1930; Peller, 1947; Greenwood, Martin and Russell, 1941). It has been reported on occasion in either sex. Thus Peller points out that a mode for the death rates in the early child-bearing ages was by no means rare in the past, and was due often to the additional hazards of child-bearing at the younger ages. Tuberculosis and other infective diseases have been causes in either sex in the past. But under modern conditions, violence is the predominant cause of a high mortality in the early twenties. I have tabulated the deaths from all causes, from violence and from all causes other than violence in Table VI. It will be seen that the violence death rate for males has a mode in the age group twenty to twenty-four years; so does that for the females, but on a rather reduced scale.

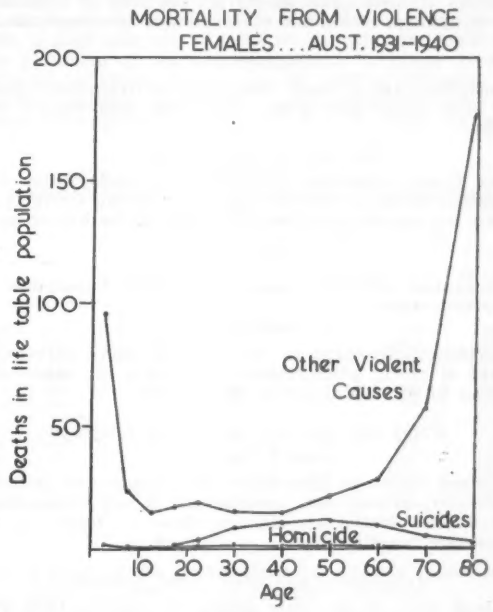

FIGURE IIB.
The deaths from violence in the life table populations, Australia, 1933 (females).

The maximum is so pronounced for the males, however, that it also causes a maximum in the male mortality rate from all causes; but for the female there is no maximum for the death rates at the same age. When the violence deaths are subtracted out of the mortality, there is a constant rise in the mortality from all other causes from the ten to fourteen years age group onwards.

Mortality by Types of Violence.

In Table VII the death rates from violence have been analysed according to the type of violence for the latest completed decennium available, 1931 to 1940. Only the rates for the adult years are discussed in this section, for Dr. F. W. Clements (1952) has considered the mortality of children from violence elsewhere. Each of the periods that I am considering is complicated by some special feature, and in discussing violence it is well to remember that the early years of the decennium 1931 to 1940 were years of economic depression; thus industrial accidents and automobile accidents may have been expected to be lower than they would have been in the absence of a depression. Owing to the great number of types of violence, a few salient points only brought out by Table VII and Figure II can be briefly mentioned.

Suicide.

The death rates of males from suicide tend to increase throughout life. The death rates of females from suicide are lower than those of the males, and there tends to be a maximum in the rates in the early fifties. The death rates standardized on to the 1933 life table populations are 211 per million *per annum* for males and 70 per million

per annum for females. When these are compared with the total death rates standardized onto the same populations, it may be inferred that at the rates of mortality existing in 1931 to 1940, about 1·4% of males and 0·5% of females would end their life by suicide.

Homicide.

Homicide is evidently a much less important cause of death than suicide.

Venomous Animals.

Deaths at these adult ages from the bites of venomous insects, spiders, snakes and other animals are uncommon.

Poisonings.

Poisonings due to food, inhalation of irrespirable gases and other accidental acute poisonings also caused few deaths.

Fire and Accidental Burns.

The group comprising deaths due to conflagration and accidental burns is of little importance in the active years of life, but covers a number of deaths at the higher ages.

Suffocation.

Mechanical suffocation also is of slight importance in the active years.

Drowning.

Accidental drowning is the cause of many preventible deaths of young adult males. It is again the cause of a number of accidental deaths of old men.

Mines and Quarries, Machinery, Railways and Tramways.

Violent deaths in association with mines and quarries, machinery, railways and tramways are largely occupational, and so the deaths are usually those of males in the productive years of life.

Automobiles and Other Land Vehicles.

About 2·5% of all male deaths in 1931 to 1940 were due to accidents involving automobiles and other land vehicles.

Water Vehicles and Air Vehicles.

Few deaths occurred in this period due to accidents associated with water or air vehicles. The deaths from air vehicles were usually those of young adult males, and were no doubt often associated with training.

Other Falls of Unspecified Nature.

Falls of unspecified nature were of small importance at the younger ages, but caused greater mortality among older persons, especially those over the age of seventy-five years. The mortality is probably understated, for if a fall caused chronic invalidity leading to death from bronchopneumonia after many months, then the accident might often not be mentioned on the certificate or coded by the statistician.

The death-rate for males was 1·2 and for females 2·2 per thousand *per annum* at ages over seventy-five years. This higher female rate is to be attributed to the well known frequency with which females sustain fracture of the neck of the femur.

Other Causes of Death.

Other specified causes such as accidental death by other crushings, animals (that is, traumatic rather than venomous), hunger, thirst, cold, heat, lightning, electricity and judicial execution are of less importance from a numerical point of view.

Summary.

The mortality from violence in Australia over the years 1908 to 1945 has been studied. It has been shown that violence and accidents form an important class of causes of death. In general the death rates from this class of causes have not fallen so rapidly as deaths from all other causes, although the therapy of accidents has improved greatly. It can be concluded only that the risks run with the increasing industrialization and mechanization have increased. Although the death rates from violence have not fallen so greatly relatively as other causes, their reduction has had some effect on the decline in mortality generally. Death rates from violence in general have a high masculinity, although when this class of deaths is analysed by type of violence, in some types the masculinity is found to be low. Violence has been shown to be the cause of an interesting feature of recent Australian life tables, the mode in the mortality rates in the early twenties. This mode is particularly due to accidents associated with automobiles, to drowning and to accidents due to firearms. The high mortality of the elderly, females especially, from fractures has been noted. A large field appropriate for preventive action exists.

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THE MORTALITY FROM VIOLENCE IN CHILDHOOD IN AUSTRALIA.

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IN an accompanying paper Lancaster (1952*b*) has reviewed the mortality from violence in Australia. Since deaths from accidents now occupy prime place in the causes of death of children, it was decided to study accidental deaths of children in more detail. For definitions and a general discussion of violence as a cause of death in Australia the reader is referred to Lancaster's article. The purpose of this paper is to present deaths and death rates of children from accidental causes with more detail, and to discuss the figures in relation to the practice of preventive pædiatrics.

Violence as a Cause of Death in Childhood.

Deaths and death rates are shown by sex and age for six periods in Tables II and III. These figures should be read in conjunction with those in Table IV, which show the relative importance of violence as a cause of death.

For our purposes it is not enough to study trends of total deaths from accidents; of greater importance are the trends of each of the major forms of violence. The patterns vary with each age group, so the trends are shown for infants aged under one year in Table V and for children in three age groups in Table VI.

Masculinity of the mortality is shown in parentheses in Tables V and VI.

Accidental Deaths in Infancy.

The number of deaths in any period in this age group is high. This means that death rates in infancy are the highest for the four age groups studied. However, deaths from violence constitute but a small percentage of the total deaths in this age group, owing to the large number of deaths from conditions peculiar to the first year of life, including prematurity and congenital malformations. Although death rates from violence have fallen during the last forty years, the drop has been much less than that for deaths due to non-accidental causes, especially the infectious and respiratory diseases. This accounts for the small but definite rise in the percentage of deaths due to accidents shown in Table IV.

The death rates for specific causes (Table V) show some interesting and informative trends over the years. Mechanical suffocation has been, and still is, given as the principal cause of death in this class, followed by death from excessive heat. The rates for the former have increased appreciably over the period studied, while those for the latter have shown a pronounced drop.

TABLE I.

Role of Children.	Accidents.	Deaths.
Passengers	87 (7·6%)	9 (19·6%)
Pedestrians ..	665 (60·6%)	28 (60·8%)
Pedal cyclists ..	269 (24·5%)	6 (13·0%)
Motor-cyclists ..	40 (3·6%)	1 (—)
Motor-car drivers ..	38 (3·4%)	2 (—)
Total ..	1096	46

A number of investigators (Brend, 1915; Greenwood, Martin and Russell, 1941; Davidson, 1945; Werne and Garrow, 1947; Bowden, 1950; Bowden and French, 1951; Swinscow, 1951) have studied the problem of death from so-called mechanical suffocation. In each series a large majority of the deaths were found to be due to some natural cause, usually a respiratory infection, which had been overlooked by the physician notifying the death. Greenwood and his co-authors (1941) point out that deaths from overlaying were twice as numerous on Saturday night, suggesting that drunkenness of the parents was a factor in many cases. It is doubtful if this is a factor in Australia. An error in diagnosis of this nature is likely to have serious repercussions in at least two directions. It causes unnecessary additional mental trauma to the parents, and especially the mother, who believes that owing to some fault in the management of the infant she has been directly responsible for its death. Because the error obscures the real cause of death, it interferes with the work of reducing infantile mortality.

Physicians and coroners can do much to correct this set of inaccurate data and prevent much unnecessary suffering. The former can contribute by refusing to consider mechanical suffocation as a cause of death until all other possibilities have been excluded, and the latter by declining to accept a notification for death due to mechanical suffocation, unless a post-mortem examination carried out by a skilled pathologist has failed to establish a natural cause of death.

Excessive heat as a cause of death appears to be in much the same category as mechanical suffocation. A limited number of studies have been made of the effects of excessive heat on both well and sick infants. Cardullo (1949), whose work seems to be the most comprehensive, found that the body temperature rose in the majority of infants under two years of age when they were exposed to sustained environmental heat. The temperature of the well and sick children in his series, with the exception of those afflicted with congenital or acquired cerebral injury, returned to normal shortly after the fall in atmospheric temperature, or when they were placed in a cool room. A preexisting acute or chronic infection, especially of the respiratory tract, usually meant a higher and more prolonged hyperpyrexia. In Cardullo's series, whereas all normal and otherwise sick infants recovered, a quarter of those with a cerebral defect or injury died. This is the experience of several other observers (Mader, 1922; Akerron, 1943; Urwitz, 1946).

These findings suggest that certification of death due to excessive heat is questionable. The practice of the Bureau of Census and Statistics, Australia, is to register death as due to excessive heat only if there are no facts enabling the death to be recorded in another category. Because of the apparent correlation between cerebral injury and heat, a good case can be made for an extensive inquiry by the physician to exclude even slight or minor cerebral damage in these cases. In the United Kingdom the Registrar-General refers back to the certifying doctor, for more information, death certificates in which excessive heat is given as the cause of death. This is a procedure that could with profit be employed in Australia.

TABLE II.

Deaths from Violence in Children by Age and Sex for Certain Periods.

Period.	Sex.	Deaths at Ages (Years).			
		0	1 to 4	5 to 9	10 to 14
1908 to 1910 ..	M.	356	419	266	296
1911 to 1920 ..	M.	725	1,471	1,049	1,105
1921 to 1930 ..	M.	511	1,584	1,225	1,216
1931 to 1940 ..	M.	549	1,310	1,111	1,280
1941 to 1945 ..	M.	304	670	536	581
1946 to 1950 ..	M.	355	801	522	444
1908 to 1910 ..	F.	249	360	171	119
1911 to 1920 ..	F.	534	1,152	516	312
1921 to 1930 ..	F.	500	1,039	586	401
1931 to 1940 ..	F.	454	854	485	322
1941 to 1945 ..	F.	232	364	201	148
1946 to 1950 ..	F.	226	466	224	120

Most of the remaining deaths can be put in the category of insufficient or inadequate parental (meaning usually maternal) care or supervision. This is, except on rare occasions, not deliberate; it is rather lack of thought or awareness, or inability to see the hazard as it exists for the infant. In this category we would put accidental poisoning, falls, burns and scalds and drowning. In the last five-year period these conditions were responsible for approximately 20% of the deaths due to accidents, compared with 27% in the period from 1911 to 1920.

Death rates from accidental poisoning and drowning have been more or less stationary for forty years; those from falls have actually increased. Burns, scalds and fire have taken smaller tolls of infant life in the two most recent quinquennial periods. This is probably the result of the replacement of open fires by safer appliances. With the great advances in infant welfare in Australia in the last thirty years or so, as revealed by the spectacular fall in infantile mortality rates, we should be entitled to expect a more pronounced decline in deaths from these conditions.

Surely the heading "Lack of Care of Infants" in the "International List of Deaths" is an expression of a social problem, being the ultimate stage of continuing neglect for one of the many reasons known to social welfare workers. It is disturbing to realize that this condition is still given as the cause of death of a number of infants each year. The fall in cases of infanticide and homicide is probably an expression of the easy adoption of the unwanted infant.

The masculinity of the mortality from various causes shows some curious unexpected figures. Why there should be a predominance of female deaths due to traffic accidents, accidental poisoning and falls is hard to explain. Almost all infants involved in traffic accidents would be accompanied by an adult and would not be wanderers on the street, as in the next age group.

Accidental Deaths of Children Aged One to Fourteen Years.

Although there has been a fall in the death rates from accidents in the three age groups mentioned, the percentages of deaths from this cause have risen sharply during the five periods studied. This is due to the precipitous decline in non-accidental deaths and the much smaller fall in accidental deaths.

Accidents from Road Vehicles, Water Transport and Aircraft.

Accidents from road vehicles, water transport and aircraft represent the most important cause of death in this class in the three age groups in both sexes. Of the various forms of transport, automobile accidents predominate. The rates which increased to peaks in the 1931-1940 decennium in most sex-age groups have declined since. The one exception is the rate for males aged one to four years; these have increased in the last quinquennial periods. The most pronounced declines are in the males and females aged ten to fourteen years. These falls in rates may well be the outcome of the educational campaigns on road safety conducted by special police squads and other groups. The rises in death rates in males aged one to four years and the relatively insignificant fall in the other groups prompt the question whether school educational campaigns should be augmented by more intense instruction by parents at home, about the hazards of traffic. This may call for different kinds of techniques from those used at school. It will require the firm conviction of all parents about the hazards that await their children. In other words, group instruction of all parents may be a necessary preliminary step.

TABLE III.

Death Rates from Violence in Australia.

Period.	Sex.	Deaths from Violence per Million Years of Life Experienced at Ages (Years).			
		0	1 to 4	5 to 9	10 to 14
1908 to 1910 ..	M.	2,035	708	407	481
1911 to 1920 ..	M.	1,079	627	398	463
1921 to 1930 ..	M.	799	628	394	409
1931 to 1940 ..	M.	947	569	366	402
1941 to 1945 ..	M.	817	532	387	403
1946 to 1950 ..	M.	793	499	322	321
1908 to 1910 ..	F.	1,519	639	264	195
1911 to 1920 ..	F.	837	508	198	131
1921 to 1930 ..	F.	768	428	194	137
1931 to 1940 ..	F.	798	387	166	104
1941 to 1945 ..	F.	648	301	151	106
1946 to 1950 ..	F.	531	304	142	95

The reports of the various State departments of road transport indicate that road accidents involving children are of four types: (*a*) those in which the child is a passenger in the vehicle and thus not responsible for the accident; (*b*) those in which the child is involved as a pedestrian; (*c*) those in which the child is involved as a pedal cyclist; (*d*) those in which the child is the rider of a motor-cycle or the driver of a motor-car.

We should expect the majority of the victims in group (*d*) to be young adolescents. Unfortunately, for a reason that is not apparent, the figures giving the details of traffic accidents issued by the various authorities, including the Commonwealth Statistician, are not given in the same age groups as those used in *Demography*. Until this is done it is not possible to collate road accidents with accidental deaths.

The number and percentages of children aged under seventeen years involved in road traffic accidents in the fiscal year 1950-1951 in New South Wales are as shown in Table I.

Accidental Poisoning.

Accidental poisoning is a significant cause of death in the one to four years age group, but claims relatively few victims in the older age groups. Ryan (1952), in his study of accidental poisoning of Queensland children, found a wide range of domestic and pharmaceutical products responsible. It seems that to the ordinary household and farm commodities, which are hazards to the inquiring mind of the young child, must be added an ever-increasing range of pharmaceutical products. Many of these are not harmful to adults in the prescribed dosages, but can be fatal to infants and young children. Ferrous sulphate pills are in the category, having been incriminated by several investigators as the cause of death of a number of young children (Thompson, 1950; Spencer, 1951; Holzel and James, 1951). Even the ubiquitous aspirin has been responsible for non-fatal poisoning of a number of infants (Ryan, 1951; Holzel and James, 1951). The mortality is about twice as high for males, this being apparently an example of greater curiosity.

Parents must accept a considerable share of the responsibility for accidental poisoning of their children; but some of this must lie with those whose task it is to interpret to parents the principles of preventive medicine. Too frequently parents do not think that a tablet that can be freely taken by adults should be securely locked away from children.

TABLE IV.

Relative Importance of Violence as a Cause of Mortality in Children.

Period.	Sex.	The Ratio of Violent Deaths and All Deaths Given as a Percentage at Ages. (Years.)			
		Under 1	1 to 4	5 to 9	10 to 14
1911 to 1920 ..	M.	1·4	8·5	17·8	27·5
1921 to 1930 ..	M.	1·2	11·4	23·0	29·2
1931 to 1940 ..	M.	2·1	14·9	24·1	32·8
1941 to 1945 ..	M.	2·1	16·7	28·9	35·9
1946 to 1950 ..	M.	2·5	25·6	36·3	41·9
1911 to 1920 ..	F.	1·4	7·5	9·9	9·3
1921 to 1930 ..	F.	1·6	9·9	13·8	12·9
1931 to 1940 ..	F.	2·3	11·6	14·3	12·4
1941 to 1945 ..	F.	2·1	11·3	15·6	14·3
1946 to 1950 ..	F.	2·2	10·4	23·3	17·4

The pharmaceutical houses can certainly help with this problem. Bottles could be more clearly labelled as poisonous to children in uncontrolled doses. Also there does not seem to be any reason why tablets and pills meant for adult consumption should be sugar-coated.

Accidental Falls.

Accidental falls account for a small but fairly constant percentage of the total accidental deaths in each of the age-sex groups. The rates seem to have reached a peak in the five-year period 1941 to 1945 and to have sharply dropped in some of the age groups in the most recent period. One wonders what has been the effect on the types of injuries caused by falls, of the widespread use in the last twenty years of concrete and asphalt to seal surfaces in public places, around schools and homes.

The mortality from accidental falls is about twice as high for males as for females, being perhaps an expression of the greater spirit of exploration and more robust play.

Accidents by Fire, Burns and Scalds.

Accidents by fire, burns and scalds have been an important cause of death in the youngest age group, less so in the five to nine years of age group, and relatively insignificant in the oldest group. This type of accident generally takes place in the home and so is more likely to involve pre-school children than those in the older age groups. When behaviour becomes more clearly defined on a sex basis—that is, after the age of four years—the rates have been higher for females; before this age the reverse applied.

Deaths from these causes have fallen considerably in the forty years under review; this fall is the main factor responsible for the overall decline in mortality rates due

TABLE V.

Death Rates and Masculinity of Death Rates in Australia in Infancy.

Causes of Death.[1]	Deaths per Million per Annum.									
	Males.[2]					Females.				
	1911 to 1920.[2]	1921 to 1930.	1931 to 1940.	1941 to 1945.	1946 to 1950.	1911 to 1920.[2]	1921 to 1930.	1931 to 1940.	1941 to 1945.	1946 to 1950.
Accidents from motor vehicle, water transport, aircraft (800 to 866) ..	—	27 (158)	36 (92)	19 (70)	13 (61)	—	17	39	27	21
Accidental poisoning (870 to 888)	21 (150)	9 (64)	14 (66)	8 (72)	11 (91)	14	14	21	11	12
Accidental falls (900 to 904)	25 (176)	13 (46)	21 (91)	22 (88)	25 (65)	14	28	23	25	38
Accidents caused by fire and hot substances *et cetera* (916 to 917) ..	125 (87)	131 (111)	105 (124)	75 (107)	72 (218)	143	117	83	70	33
Mechanical suffocation (924 to 925)	272 (103)	278 (134)	269 (114)	411 (198)	373 (174)	262	207	234	207	214
Lack of care of infants (926)	98 (118)	72 (93)	66 (150)	35 (97)	38 (180)	83	77	44	36	21
Drowning (929)	28 (73)	30 (172)	28 (560)	24 (103)	27 (225)	38	17	5	22	12
Excessive heat (931) ..	228 (144)	142 (112)	164 (104)	81 (96)	89 (189)	158	126	157	84	47
Infanticide, homicide (980 to 983)	100 (128)	97 (98)	100 (149)	85 (100)	20 (123)	78	98	67	59	33
Other accidents	122	98	145	107	125	125	68	127	106	101

[1] The classification of causes of death used here is the 1948 Revision of the International List of Deaths. The numerals are from that list.

[2] Not given as a separate classification in this period.

[3] The masculinity of death rates is shown in parentheses. The masculinity of the death rate is equal to 100 times the male death rate divided by the female death rate.

TABLE VI.

Death Rates and Masculinity of Death Rates in Australia in Childhood.

Causes of Death.[1]	Deaths per Million per Annum.									
	Males.[2]					Females.				
	1911 to 1920.	1921 to 1930.	1931 to 1940.	1941 to 1945.	1946 to 1950.	1911 to 1920.	1921 to 1930.	1931 to 1940.	1941 to 1945.	1946 to 1950.
One to Four Years.										
Accidents from road vehicles, water transport and aircraft (800 to 866)	54 (125)	84 (150)	101 (129)	129 (230)	153 (228)	43	56	78	56	67
Accidental poisoning (870 to 888)	41 (146)	42 (168)	37 (168)	37 (194)	30 (187)	28	25	22	19	16
Accidental falls (900 to 904)	30 (176)	29 (207)	23 (134)	36 (257)	24 (218)	17	14	16	14	11
Accidents caused by fire, hot substances *et cetera* (916 to 917)	226 (89)	224 (119)	169 (113)	111 (127)	71 (105)	252	187	149	87	67
Drowning (929)	138 (205)	131 (201)	120 (179)	141 (236)	139 (323)	67	65	67	59	43
Other accidents	138	118	119	78	82	101	81	54	66	100
Five to Nine Years.										
Accidents from road vehicles, water transport and aircraft (800 to 866)	93 (211)	151 (218)	162 (234)	159 (230)	154 (226)	44	69	69	69	68
Accidental falls (900 to 904)	27 (300)	26 (260)	16 (177)	30 (230)	23 (209)	9	10	9	13	11
Accidents caused by fire, hot substances *et cetera* (916 to 917)	23 (18)	21 (45)	10 (37)	8 (38)	7 (50)	71	46	27	21	14
Drowning (929)	157 (461)	123 (492)	108 (385)	119 (424)	90 (391)	34	25	28	28	23
Other accidents ..	98	73	70	71	48	42	41	41	23	26
Ten to Fourteen Years.										
Accidents from road vehicles, water transport and aircraft (800 to 866)	85 (425)	131 (334)	169 (338)	140 (368)	109 (286)	20	39	50	38	38
Accidental falls (900 to 904)	44 (550)	35 (437)	22 (440)	52 (472)	30 (333)	8	8	5	11	9
Accidents caused by fire, hot substances *et cetera* (916 to 917)	4 (12)	8 (32)	8 (80)	10 (90)	1 (25)	33	25	10	11	4
Accidents by firearms (919)	59 (737)	32 (800)	25 (833)	33 (3300)	24 (2400)	8	4	3	1	1
Drowning (929)	159 (454)	124 (343)	105 (456)	110 (323)	89 (386)	35	36	23	34	23
Other accidents	116	87	81	69	69	27	25	13	11	20

[1] The classification of causes of death used here is the 1948 Revision of the International List of Deaths. The numerals are from that list.

[2] The masculinity of death rates is shown in parentheses. The masculinity of the death rate is equal to 100 times the male death rate divided by the female death rate.

to accidents. The change from open fireplaces and fuel ranges to electric and gas appliances is probably one cause of this fall. To this, perhaps, should be added the more satisfactory treatment of burns evolved in the last ten years or so.

Accidents due to Firearms.

Accidents due to firearms affect males almost exclusively. Rates have fluctuated, being particularly high in the decennium 1911 to 1920—that is, during a period when a high percentage of the population lived in the country.

Drowning.

Drowning accounts for a high percentage of the deaths due to accidents in the three age groups, being the second most important cause. The rates are higher for males. Until the most recent quinquennial period the rates have been fairly constant; a fall is recorded in all age groups except that of males aged one to four years.

Discussion.

The total mortality rates in the age-sex groups studied have fallen considerably in the last twenty-five to thirty years, owing mainly to the sharp decline in deaths due to infectious diseases (Lancaster, 1952*a*). The importance of accidental deaths as a cause of death of children has thus been emphasized. If total mortality rates for children are to be reduced still further, there must be a considerable reduction in deaths due to accidents. Dietrich (1950) has pointed out that this calls for different techniques from those which have proved so successful in the control of infectious diseases.

However, we do need to know a great deal more about the factors responsible for accidents to children. A study designed to collect such information is in hand at the moment. When we are armed with this information, the next step will be the formulation of programmes of prevention.

A good start has already been made in the campaign against traffic accidents; but, as has been pointed out above, the educational campaign must be extended to pre-school children at home.

Summary.

The mortality from accidents in childhood in Australia over the years 1908 to 1950 has been studied. It has been shown that accidents are the most important cause of death in the age groups one to fourteen years. Whereas death rates from non-accidental causes have fallen by about 70%, deaths from accidents have fallen by less than 30%. Accidental deaths in infancy do not form a high percentage of deaths in this age group, because of the high death rates from conditions peculiar to the first year of life. Two conditions given as causes of death in this age group—namely, mechanical suffocation and excessive heat—require further study.

In the three age groups one to four years, five to nine years and ten to fourteen years, traffic accidents are the most important cause of death. Accidental poisoning is important in the one to four years age group, while drowning claims a significant number of deaths in the three age groups.

If death rates in childhood are to be lowered significantly in the future, this will be achieved only by a considerable reduction in the incidence of all forms of accidental death.

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ANAEROGENIC PARACOLON BACILLI ASSOCIATED WITH GASTRO-ENTERITIS IN CHILDREN.

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MANY papers have been published on the various groups of organisms which are believed to be the ætiological agents of gastro-enteritis, the best known being those of the *Shigella* and *Salmonella* groups, and to a lesser extent *Proteus*, *Ps udomonas pyocyanea* and *Staphylococcus aureus*. In recent years organisms of the *Paracolobactrum* group have received attention as possible pathogens. A review of the literature over the past fifteen years reveals that the earlier workers, for example, Sandiford (1935), believed that paracolons were of no pathogenic importance. Later workers have reported outbreaks of infantile diarrhœa and gastro-enteritis which were believed to have been caused by paracolon bacilli—for example, Neter and Clark (1944), Stuart and van Stratum (1945), Sevitt (1945), Berger (1945), Edwards (1945), Ferguson and Wheeler (1946). Others who have written on the pathogenicity of this group, not necessarily in children, include Rhodes (1942), Cope and Kilander (1942), Sachs (1943), Stuart and Rustigian (1943), Barnes and Cherry (1946), Stuart *et alii* (1946), Plass (1947), Christensen (1947), and Mushin (1949*a*).

From a study of these works the role of the paracolon group of organisms in the intestine has been ably summarized by Neter and Clark (1944), who state that paracolon bacilli (i) may be saprophytes encountered in normal individuals, (ii) may be present in the intestinal tract as harmless organisms in patients with diarrhœal disease caused by enteric pathogens or other incitants (parenteral infection), (iii) may be associated with enteric pathogens and play a secondary part as incitants of diarrhœa, and (iv) may be a primary cause in certain susceptible individuals, particularly infants and children. These four points, together with the difficulties associated with the classification of the group, since it comprises so many sero-

logically and biochemically heterogeneous types, make the paracolon bacilli a problem for bacteriologists.

It is proposed to discuss in this article strains of anaerogenic paracolon bacilli which were isolated at the Adelaide Children's Hospital from the fæces in 13 cases of gastro-enteritis in young children, and which are believed to be the ætiological agents of the disease in these particular cases.

Methods of Isolation.

The routine method of preparing cultures from fæces in this laboratory is to plate them directly onto Leifson's desoxycholate citrate agar, and to put a heavy inoculum into tetrathionate broth, and 10% salt meat medium for the isolation of *Staphylococcus aureus* (Maitland and Martyn, 1948). After overnight incubation, non-lactose fermenting colonies on the Leifson's plate are subcultured into Kligler's iron agar (Difco). The tetrathionate broth is plated onto SS agar (Difco), and a small inoculum of the salt meat medium is stroked onto a Löffler's blood serum slope. On the third day non-lactose fermenting colonies on the SS agar are subcultured into Kligler's iron agar. The particular group of paracolon bacilli which is being discussed, and which for convenience is known in this laboratory as the A.C.H. group, gives the same reactions in Kligler's iron agar as the *Shigella flexneri* group, that is, a red alkaline slope, and a yellow acid butt with no gas or sulphide.

Six of the 13 strains of the A.C.H. group of organisms were isolated both from Leifson's medium by direct plating and from SS agar subcultured from tetrathionate broth, five from Leifson's medium only, and two from SS agar only.

Morphology and Growth Characteristics.

The organisms are Gram-negative, non-sporing rods similar in size and shape to other members of the bacterium group. They are non-motile at 37° C, but actively motile at 22° C. They do not spread on 1% agar at 37° C. but spread very slightly at 22° C. They grow readily on ordinary media; and after twenty-four hours at 37° C. the colony on nutrient agar is small, round and translucent, with an entire edge and a convex smooth surface. The organisms grow well on Leifson's or desoxycholate citrate agar, forming small colourless colonies similar in size and shape to those of the *Shigella flexneri* group; but unlike this group of organisms, they also grow well in tetrathionate broth. The colonies on SS agar are more opaque and a little larger than those on desoxycholate citrate agar.

Biochemical Reactions.

The 13 strains of the A.C.H. organisms were all biochemically identical except that one strain gave a positive response to the methyl red test.

Fermentation reactions were carried out in 1% carbohydrate solutions in 1% peptone water, Andrade's indicator being used. The organisms fermented glucose, mannose and glycerol after twenty-four hours' incubation, and saccharose after three days' incubation, producing acid but no gas. They failed to ferment lactose, mannite, *l*-arabinose, dulcitol, inositol, rhamnose, cellobiose, galactose, salicin and maltose after one month's incubation. They did not ferment lactose after five weeks' incubation, 10% lactose agar slopes being used with brom-cresol-purple for an indicator, as recommended by Chilton and Fulton (1946). The reaction in maltose was repeated many times under different conditions, both Seitz filtered and autoclaved sugar solutions being used, but they failed to ferment it even after five weeks' incubation. Citrate could be utilized as the only source of carbon, growth being more rapid at room temperature than at 37° C. The methyl red test result was negative with all strains isolated, apart from one "Carpenter" strain, which gave a positive reaction. Acetyl-methyl-carbinol was not produced by any of the strains. Indole was produced by all. Gelatin was not liquefied. Nitrates were reduced to nitrites. Sulphide was not produced either in Kligler's agar or in 1% tryptone water and lead acetate paper after one month's incubation. With the medium recommended by Schneider and Gunderson (1946) the organisms did not split urea.

The A.C.H. paracolon group is biochemically similar to *Proteus rettgeri* except that it does not hydrolyse urea, and therefore must be placed in the *Paracolobactrum* group rather than in the *Proteus* group (Breed *et alii*, 1948). Biochemically the organisms are identical with two of the Sachs (1943) types, B81 and B105, the anaerogenic paracolon type 29911 (Stuart *et alii*, 1943), the *Bacillus wakefield* (Berger, 1945), and MacLennan's type P25 (1945). In 1946 Wheeler and Stuart showed that the *Bacillus wakefield* belonged to the same group as paracolon type 29911, and in 1946 Stuart *et alii* proved that the Sachs types B81 and B105 were also members of this group.

Serological Investigations.

A complete study of the serology of these organisms was not undertaken, but an attempt was made to determine (i) whether the 13 strains of the A.C.H. group of paracolons were identical, (ii) whether there was any relationship between these organisms and those of the *Shigella* or *Proteus rettgeri* groups, and (iii) whether there was any relationship to the biochemically similar anaerogenic paracolon type 29911.

To Determine Whether the 13 Strains of the A.C.H. Group Were Identical.

An "O" antiserum was prepared against the first strain isolated, that is, the "McCann" strain, and the titre of the antiserum against its homologous organism was 1:640.

Any organisms which were subsequently isolated and suspected of being in the A.C.H. group, were tested by the slide agglutination method against this serum, and in this way each of the other 12 strains was detected. Confirmatory tube agglutinations were then performed, the tests being carried out by heating in a water-bath at 55° C. for two hours and then leaving overnight at room temperature. Each of the other 12 strains agglutinated the McCann "O" antiserum to its full titre of 1:640, with the exception of one strain "Carpenter", which agglutinated it to 1:320. Agglutinin absorption tests were then performed against each strain. The serum was first diluted 1:10, and absorption was carried out at 55° C. for two hours in a water-bath, then overnight in a refrigerator. The absorbed serum was then tested against each of the 13 strains. With the exception of the Carpenter strain, all the other strains had removed the McCann agglutinins completely, whereas the Carpenter strain had only reduced the titre from 640 to 200, which showed that this organism possesses only some of the somatic antigens of the McCann strain.

An "O" antiserum of the Carpenter organism was then prepared, and the titre was 1:1280. Agglutination tests with this antiserum were carried out against the other 12 strains, all of which agglutinated it to a titre of only 1:10. Agglutinin absorption tests with each of the strains showed that the major Carpenter agglutinins had not been removed, as the original titre was not reduced.

These results showed that 12 of the strains had similar "O" antigens, and one strain "Carpenter" belonged to another type sharing some of the "O" antigens of the other 12 strains.

Flagellar or "H" antisera were prepared against both McCann and Carpenter strains, formolized broth cultures which had grown at room temperature for forty-eight hours and were actively motile being used for injections. The titre of the McCann antiserum against its homologous "H" suspension was 4096 and that of the Carpenter antiserum 1024. "H" agglutination tests were performed on each of the 13 cultures against both antisera. The tests were carried out by incubating the tubes in a water-bath at 55° C. and reading the titres at the end of two hours' incubation. All strains agglutinated both the McCann and Carpenter "H" antisera to their full titres. The McCann "H" antiserum was absorbed with washed, packed Carpenter "H" organisms which had been obtained by centrifuging 1500 millilitres of actively motile broth cultures grown at room temperature and then formolized. In the same way the Carpenter "H" antiserum was absorbed with McCann "H" organisms. Cross-agglutination tests proved that in

both cases all the homologous agglutinins had been removed and therefore the "H" antigens of the McCann and Carpenter strains were identical.

From these results it can be said that of the 13 strains isolated, 12, henceforth called the McCann type, are identical, and one strain, "Carpenter", belongs to a closely related type.

To Determine the Relationship between the A.C.H. Paracolon Group and the Shigella and Proteus Rettgeri Groups.

Slide agglutination tests were performed with the McCann and Carpenter "O" organisms against antisera of the *Shigella flexneri* group, types I to VI, the Shiga type, the Boyd types I, II and III, and the Schmitz type, which were obtained from the Commonwealth Serum Laboratories of Australia. The results of these tests were all negative. However, the results of slide agglutination tests in which *Shigella flexneri* type II organisms were used against both McCann and Carpenter "O" antisera were positive. Confirmatory tests were carried out with the tube technique, and under these conditions both the antisera agglutinated this organism to a titre of 1:40. When these antisera were absorbed with *Shigella flexneri* type II organisms the homologous titres were not reduced. This showed that there was no specific antigenic relationship between the A.C.H. paracolons and *Shigella flexneri* type II organisms.

Non-Specific Agglutination of Shigella Flexneri Type II Organisms by A.C.H. Paracolon Antisera.—In 1945 Berger demonstrated an antigenic relationship between the *Bacillus wakefield* and the *Shigella flexneri* group, a relationship which Bridges and Taylor (1946) proved to be due to the non-specific α antigen of Stamp and Stone (1944). The α antigen is non-specific and is common to some organisms of the *Paracolobactrum*, the *Shigella* and the *Salmonella* groups. It produces an "O" type of agglutination and is completely destroyed by heating at 100° C. for fifteen minutes, or by treating with alcohol at 65° C. for sixty minutes. In 1949 Mushin (1949*b*) demonstrated another non-specific thermolabile antigen, known as the β antigen, which is common to certain paracolon bacilli and *Shigella flexneri Y* strains, and one of the main characteristics of which is the rapid floccular agglutination which is complete in thirty minutes at 52° C. It seemed possible, therefore, that the agglutination which we had obtained, using the McCann and Carpenter "O" antisera and the *Shigella flexneri* II organisms, was due to one of these non-specific antigens. A suspension of *Shigella flexneri* type II organisms was prepared, and one portion was heated in a boiling-water bath for fifteen minutes, another was treated with alcohol and then heated at 65° C. for sixty minutes, and a further portion was used as a control. Agglutination tests, in which these three suspensions were used against both the A.C.H. paracolon antisera, showed that the antigen had been completely destroyed both by the boiling and by the alcohol treatment, and the agglutination given with the control suspension was of a typical granular "O" type, and not floccular, nor as rapid in forming as the β type of agglutination. In view of these results it seems that the agglutination which we obtained was due to a non-specific antigen similar to the α antigen of Stamp and Stone, and that there is no true antigenic relationship existing between the A.C.H. paracolons and the *Shigella flexneri* group.

Tests with Proteus Rettgeri Strains.—As the biochemical reactions of the A.C.H. paracolon group differed from those of the *Proteus rettgeri* group only in their failure to hydrolyse urea, tube agglutination tests were performed, "O" and "H" McCann and Carpenter antisera being used against "O" and "H" suspensions of seven strains of *Proteus rettgeri* which were obtained from the National Collection of Type Cultures, London. The strains used were KB5, KB119, KB357, KB683, KB712, KB772 and JB1893, and all gave negative results, which shows that there is no antigenic relationship between the A.C.H. paracolon group and the strains of *Proteus rettgeri* which were used for the tests.

To Determine the Relationship of the A.C.H. Paracolon Bacilli to the Anaerogenic Paracolon Type 29911 Group.

Cultures of four strains of paracolon type 29911 were kindly provided by Dr. C. A. Stuart, of Brown University (United States of America). The strains were EEB, 2634, 16112 and 508. In addition, cultures of the two Sachs types, B81 and B105, which have been proved to belong to the type 29911 group, and the biochemically similar P25 of MacLennan, were obtained from the National Collection of Type Cultures, London. Both "O" and "H" suspensions of each of these cultures were prepared and tube agglutination tests were performed against "O" and "H" McCann and Carpenter antisera. The tests were carried out by heating the tubes in a water-bath at 55° C. for two hours and leaving them overnight at room temperature. The results are shown in Table I.

TABLE I.

Suspension.	McCann Antisera.		Carpenter Antisera.	
	"O."	"H."	"O."	"H."
McCann[1]: "O"	640	—	10	—
"H"	—	4100	—	1024
Carpenter[1]: "O"	320	—	1280	—
"H"	—	4100	—	1024
EEB: "O"	4	—	64	—
"H"	—	0	—	0
508: "O"	0	—	0	—
"H"	—	0	—	0
2634: "O"	4	—	0	—
"H"	—	0	—	0
16112: "O"	4	—	0	—
"H"	—	0	—	0
B81: "O"	8	—	32	—
"H"	—	0	—	0
B105: "O"	0	—	0	—
"H"	—	0	—	0
P25: "O"	0	—	0	—
"H"	—	0	—	0

[1] Control.

It can be seen from the table that the results of all the "H" agglutination tests were negative, and the "O" agglutinations were of very low titre with the exception of those from strain EEB and the Sachs type B81, which gave titres of 1:64 and 1:32, respectively, with the Carpenter "O" antiserum. When, however, the Carpenter "O" antiserum was absorbed with EEB and B81 "O" cells, it retained its homologous titre in both cases.

It must be noted that no antisera were prepared from any of the type 29911 strains or from the Sachs types, so cross-absorption tests could not be performed.

Stuart *et alii* (1946), during their work with the type 29911 strains, found that in some cross-reactions between some strains and antisera, the findings were negative at 55° C. but positive at 37° C.; and also that in some cases in which positive results were obtained at 37° C. and the material was then incubated at 55° C., complete reversion of agglutination occurred. In view of these findings all the agglutination tests were repeated at 37° C. with both "O" and "H" McCann and Carpenter antisera, and "O" and "H" suspensions of the four strains of paracolon type 29911 and Sachs types B81, B105 and P25. The only difference was an increase in titre from 1:64 to 1:128, when an "O"

suspension of strain EEB was used against the Carpenter "O" antiserum. The results of all the "H" agglutination tests still remained negative.

In spite of the similar biochemical reactions, the low titres in the agglutination tests show that the A.C.H. paracolons cannot be included in the type 29911 group used for the tests. This does not necessarily exclude them from the whole group, since Stuart *et alii* (1943) have reported that paracolon type 29911 comprises a serologically heterogeneous group, and it is possible that the A.C.H. group would give more definite reactions with other known strains of type 29911 apart from those which were available to us. However, until further serological investigations can be made to prove the linkage between the two groups, the A.C.H. paracolon bacilli must remain a distinct group consisting at present of two closely related types.

The Development of a Thermolabile Somatic Antigen.

All the cultures were stored on agar slopes under sterile paraffin oil, and after about six months subcultures were made. Slide agglutination tests with the McCann "O" antiserum were carried out on each culture for checking purposes, and one culture, "O'Reilly", failed to give a positive agglutination. It was thought that the original culture had become overgrown by a contaminant, but after the plating and subculturing of separate colonies, it was found that the O'Reilly strain was still pure and gave the original biochemical reactions, but that the tube agglutination tests failed to give any positive results. This "O" inagglutinability suggested the interference by some surface antigen. A saline suspension was prepared and heated in a boiling-water bath for forty-five minutes. This boiled suspension then agglutinated the McCann "O" antiserum to its full titre of 1:640. This phenomenon indicated the development of a heat-labile surface antigen similar to the "L" antigen described by Kauffmann (1947). The McCann "O" antiserum was then absorbed with both boiled and unboiled O'Reilly organisms, and it was found that no agglutinins were removed by the unboiled organisms and all the agglutinins were removed by the boiled organisms; this showed that the presence of the surface antigen completely inhibited the absorption of the somatic "O" antibodies by their homologous antigens.

The O'Reilly culture was subcultured and stored as before under paraffin and was reexamined several months after. It was then found that an unboiled suspension agglutinated the McCann "O" antiserum to a titre of 1:20, although the agglutination was atypical, being of the "thready" type. The "L" antigen was therefore unstable, and the culture appeared to be reverting back to the original.

After the finding of this phenomenon in one culture, agglutination tests were performed on all the other strains, both boiled and unboiled suspensions being used. Under these conditions the eleven other strains still agglutinated the McCann "O" antiserum to the full titre of 1:640, and the Carpenter "O" antiserum to the original titre of 1:10. The boiled suspension of the Carpenter organisms, however, agglutinated the McCann "O" antiserum to 1:80 instead of 1:320 when the unboiled suspension was used; this indicated that an "O" antigen in the Carpenter organism which was common to the McCann strain was partially heat-labile, while the lesser "O" antigen possessed by the McCann organisms and common to the Carpenter strain was heat-stable.

The Pathogenicity of the A.C.H. Paracolon Group.

Each of the 13 children from whom the A.C.H. paracolon organisms were isolated were objects of definite clinical infection, having acute diarrhœa accompanied by vomiting. Nine of the children suffered for from seven to ten days, three children suffered for three weeks, and an older child of five years of age had intermittent attacks of diarrhœa and vomiting over a period of five months.

To determine whether normal children harbour the A.C.H. paracolons as members of the harmless normal flora of the intestine, specimens of fæces were examined from 100 normal children in the same age group as the infected children. The A.C.H. organisms were not isolated from any of the specimens examined.

Samples of serum from 11 of the 13 children affected were examined for antibodies, "O" and "H" suspensions of both the McCann organism and the organism isolated from the child being used. The two children who were not tested were discharged from hospital before blood could be collected, and one of these was unfortunately the McCann child from whom the first isolation was made. The blood was collected fourteen days after the onset of the symptoms if possible. Positive results from "O" agglutination tests were obtained with each of the 11 sera, but the results of all the "H" agglutination tests were negative.

The date of collection, the ages of the children, and the agglutination titres are shown in Table II.

TABLE II.

Date.	Identification.	Age of Child.	Suspension Used.	
			McCann "O".	"O" Suspension of Child's Own Organism.
9. 1.45	McCann.	1 year 11 months.	—	—
8. 5.45	Byrne.	1 year 7 months.	—	—
22. 6.45	Boodnikoff.	6 weeks.	64	64
7. 2.46	Page.	4 months.	32	32
20. 2.47	Hamdorf.	4 years 5 months.	64	64
24. 3.47	Carpenter.	1 year 11 months.	32	128
26. 4.47	O'Reilly.	11 months.	16	16
20. 5.47	Turner.	5 years.	32	32
22. 5.47	Treaize.	1 year 6 months.	128	128
15. 3.49	Bester.	10 months.	128	128
24. 4.49	Nuske.	6 months.	32	32
19. 6.50	Mitchell.	4 months.	16	16
24.10.51	Currie.	4 months.	128	128

It is interesting to note that the child of only six weeks of age developed agglutinins to a titre of 1:64, and also that of the 13 children 11 were under two years of age. In each case it will be seen that the titre obtained with the organism isolated from the child was identical with that obtained with the McCann "O" suspension, with the exception of the Carpenter child, whose titre with its own organism was 128, and with the McCann suspension only 32. It was this finding which led to further investigations and the conclusion that the organism was of a different type from the McCann organism. At the time when these agglutination tests were performed the O'Reilly organism had not developed any "L" antigen.

The opportunity arose of carrying out a series of agglutination tests over a period of time on the Bester child, who was ten months old when first infected. The first specimen of serum was tested fourteen days after the onset of the diarrhœa, and the titre against an "O" suspension of its own organism and the McCann organism was 1:64. One week later, when the child was recovering, the titre had risen to 1:128. About two months later the titre had fallen to 1:16, and about one year later no agglutinins were detected. This rise in antibody titre after the isolation of the organism, the gradual fall after the recovery of the child, and finally the disappearance altogether of the agglutinins provide further evidence of the pathogenicity of this group of paracolon bacilli.

To show that the children were not infected by one of the known pathogens of the *Shigella* or *Salmonella* group which had not been isolated from the fæces, the sera from the 11 children were also tested for agglutinins of these two groups, and all yielded negative results.

In addition, to prove that normal children do not possess the A.C.H. paracolon agglutinins as natural antibodies in the blood, the sera from 100 normal children were collected and tested against an "O" suspension of the McCann organisms. Samples of blood were collected for the tests from children in each of the age groups of the infected children. No positive agglutination test results were obtained.

There still remained the possibility that children with *Salmonella* or *Shigella* infections developed antibodies common to either of those groups and the A.C.H. paracolons, especially as many other workers, for example, Stuart *et alii* (1943), Berger (1945), Taylor and Bridges (1946), Ferguson and Wheeler (1946) and Young (1946), have demonstrated antigens which are shared by paracolon bacilli and organisms of the *Shigella* and *Salmonella* groups. If this were so, then it was possible that the children whom we claimed to have been infected by the A.C.H. paracolon organism were actually infected by known pathogens which we had failed to isolate. Examination of sera from 25 subjects of *Salmonella* infections, 10 subjects of *Shigella flexneri* type II infections, and five subjects of *Shigella sonnei* infection, all of whom showed an antibody response to their own specific organisms, failed in all cases to demonstrate agglutination of an "O" McCann suspension.

Since every child from whom the A.C.H. paracolons were isolated, and whose serum was tested, showed an antibody response, and since none of 100 normal children yielded positive results from either fæcal culture or blood agglutination tests, and in view of the clinical condition of the children at the time of isolation of the organisms, we consider that it is justifiable to assume that this group of paracolon bacilli is capable of causing mild gastro-enteritis in young children.

Discussion.

A review of the literature dealing with the pathogenicity of the paracolon bacilli showed that many of the workers have based their reasons for pathogenicity only on such evidence as the isolation of a particular paracolon organism, its predominance over other organisms present on the culture, the clinical condition of the patient, the failure to isolate any known pathogen, the fact that paracolons are more often isolated from subjects of gastro-enteritis than from normal persons, and the isolation of the suspected paracolon organism from persons handling the food of the patients.

In our opinion the finding of specific agglutinins in the blood is additional evidence for the pathogenicity of any intestinal organism. In 1945 Draper pointed out that the agglutination test could be used as a diagnostic aid in cases of gastro-enteritis in children caused by organisms of the *Salmonella* and *Shigella* groups. He found that of 25 subjects of *Salmonella* infection, 18 developed *Salmonella* agglutinins, and of 24 subjects of *Shigella flexneri* type II infection, 22 developed *flexneri* agglutinins, while each of four subjects of *Shigella sonnei* infection developed *sonnei* agglutinins. He also found that these agglutinins were not among the natural antibodies found in the blood of normal children.

Some workers on paracolon bacilli have tried without success to demonstrate the presence of specific antibodies in the serum of their patients, for example, Sandiford (1935), Ferguson and Wheeler (1946) and Herta Schwabacher (1949). Other workers have found that in a large percentage of their cases the patients developed agglutinins against the paracolon strains isolated from the fæces. Rhodes (1942) found that sera in his five cases yielded positive agglutination test results. In 1943 Sachs demonstrated the presence of agglutinins against the Sachs strains in 62 sera which he tested, and of these two were type B81 and two were type B105. He found also that 200 samples of sera from normal persons gave no reaction. In 1945 Sevitt found that the sera of nine of 16 patients (12 of whom were under six months of age) yielded positive agglutination test results, while seven yielded negative results. Berger (1945) showed the presence of agglutinins in the serum of a patient from whom the *Bacillus wakefield* was isolated, and Mushin (1949*a*) demonstrated agglutinins in the blood of persons from whom the sulphide-forming, gas-producing *Paracolon melbourne* was isolated, but failed to detect any antibody response to other paracolon strains isolated.

It is thus interesting to note that the articles dealing with the anaerogenic paracolon bacilli, Sachs's types B81 and B105, and *Bacillus wakefield* contain reports of positive agglutination test results to support claims for pathogenicity.

From a survey of the reports on the isolation of anaerogenic paracolon bacilli, and from the evidence that has been presented for their pathogenicity, it seems that this section of the paracolon group does play an important part in the ætiology of gastro-enteritis and diarrhœa in many parts of the world. It is true that much of the evidence is circumstantial, but that does not mean that it should be disregarded. It must be remembered that laboratories acting as centres for examining cultures sent from various parts of the country are at a definite disadvantage in relation to collecting samples of blood from the patient, whereas hospital workers are able to follow up their cases more easily.

A brief summary of some of the work that has been performed on anaerogenic paracolon bacilli will give some idea of the distribution and significance of these organisms.

In 1943 Sachs reported the isolation in India and Egypt of 107 strains of "non-mannitol fermenting bacilli", which included his types B81 and B105. The cultures were obtained from patients in clinical cases of dysentery, and as was pointed out earlier, positive agglutination test results were obtained from each of 62 samples of serum. No isolations were made from 15,903 specimens of fæces from food-handlers.

In 1943 Stuart *et alii* reported the isolation of type 29911 paracolon bacilli, which they believed to be pathogenic, and later in 1946 they studied 109 cultures isolated in Connecticut from subjects of gastro-enteritis. Only one isolation was obtained from 300 normal persons. In 1945 MacLennan investigated 310 cultures of "non-mannite fermenting dysentery bacilli" which were isolated in the Middle East, and which included Sachs's types B81 and B105, as well as MacLennan's type P25. All these organisms were isolated from cases of dysentery or diarrhœa.

In England, Berger (1945) isolated the *Bacillus wakefield* from an outbreak of diarrhœa among children in a nursery at Boston Spa, from a case of gastro-enteritis, and also from an adult with recurrent diarrhœa.

Plass, from Minneapolis, in 1947 reported 80 cases of food poisoning from an army officers' mess. Paracolon bacilli similar to type 29911 were isolated from the fæces of 13 of 16 patients who required admission to hospital, and also from the fricassee of chicken which had been consumed by all the affected persons.

In 1947 Galton *et alii* reported on the distribution of type 29911 paracolon bacilli in Florida. One hundred and fifty-eight cultures were obtained from 31 different cities, but apart from two isolations, one of which was from a two-year-old child with diarrhœa of two weeks' duration, there were no case histories available, and hence it was impossible to assess their significance in the community.

Many workers have noted a definite increase in the number of paracolons isolated from subjects of gastro-enteritis whether known pathogens have been found or not, and similarly it has been the experience in this laboratory to find that aerogenic paracolons are widespread in the fæces of both normal children and those suffering from gastro-enteritis, with a much higher percentage of isolations from the subjects of gastro-enteritis. However, we have found from agglutination tests carried out on a number of samples of serum from children from whom these aerogenic paracolons have been isolated, that there was no antibody response, which indicated that these particular aerogenic strains formed part of the normal flora of the intestine. We believe that the increase in aerogenic paracolons in cases of gastro-enteritis is due to the abnormal condition of the intestine, which, in its state of lowered resistance, allows organisms to thrive and multiply, and possibly to aggravate the existing damage, although normally these organisms would be unable to become well established.

It is possible that infection by these organisms is overcome successfully by older children and adults. If these people do become infected, the infection, although too mild

to require medical treatment, is sufficient to keep the organism circulating within the community. The fact that strains isolated in 1951 were biochemically and serologically identical with strains isolated in 1945 supports this suggestion.

As only 13 isolations of the A.C.H. group of paracolon bacilli were made over a period of nearly seven years, this group can scarcely be considered a major cause of gastro-enteritis in South Australia, but at the same time its presence in the community is a potential source of danger to the infant population, particularly in maternity hospitals and children's institutions.

Summary.

1. A report is given on a group of anaerogenic paracolon bacilli called, for convenience, the A.C.H. paracolon group. These organisms were isolated from the fæces in 13 cases of gastro-enteritis in young children, all of whom were patients at this hospital. Similar organisms were not isolated from the fæces of 100 normal children in the same age group.

2. The group consists of two types, namely, the "McCann" and "Carpenter" types.

3. Twelve of the strains belonged to the "McCann" type and were biochemically and serologically identical. The one "Carpenter" type differed biochemically in giving a positive response to the methyl red test, and in certain serological reactions. In the major biochemical reactions the A.C.H. group is similar to the anaerogenic paracolon type 29911 organism of Stuart *et alii* (1943).

4. Agglutinin absorption tests show that the "McCann" and "Carpenter" types have identical "H" or flagellar antigens and that some "O" or somatic antigens are common to both types.

5. Agglutination tests showed that the agglutinating sera of both A.C.H. types contained a non-specific aglutinin which was responsible for the agglutination of *Shigella flexneri* type II organisms. This agglutination was probably caused by a non-specific antigen present on the *Shigella flexneri* type II organisms and similar to the a antigen of Stamp and Stone.

6. The A.C.H. group of paracolon organisms did not show any definite serological relationship with four strains of type 29911 paracolon bacillus, Sachs's types B81 and B105, and MacLennan's P25.

7. Sera collected from 11 of the patients agglutinated the "O" suspensions of both the McCann organism and the organism isolated from the particular child. All "H" agglutination test results were negative. Results of agglutination tests with *Salmonella* and *Shigella* organisms were negative. Sera from 100 normal children in the same age group failed to agglutinate the McCann "O" suspension. Sera from 25 patients with *Salmonella* infection and 15 with bacillary dysentery failed to agglutinate the "O" suspension of the McCann organism, but all agglutinated suspensions of their own particular organisms.

8. Serial agglutination tests on the serum from one child showed a rise and fall of titre as the severity of the infection receded.

9. A brief review of the literature, dealing with the pathogenicity of the anaerogenic paracolons in various parts of the world, is given.

Conclusion.

The hypothesis that the A.C.H. group of paracolon bacilli can cause mild gastro-enteritis is supported by the isolation of these organisms only from subjects of gastro-enteritis, and also by the presence of specific agglutinins of the group in the sera of the patients, whereas these organisms and agglutinins were absent from the fæces and sera of normal children and from children with known *Salmonella* and bacillary dysentery infections.

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I wish to thank Mr. F. Draper, B.Sc., for the wholehearted cooperation and advice he has given throughout this work, and also Dr. C. A. Stuart, of Brown University, Providence, Rhode Island, United States of America, for kindly providing us with four strains of the type 29911 paracolon bacillus.

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Reviews.

MICROBIOLOGY FOR NURSES.

"MICROBIOLOGY: WITH APPLICATIONS TO NURSING", by Catherine Jones Witton, is a book clearly set out, with many excellent diagrams and informative photographs.[1] It is divided into seven sections: "The Basic Structure and Activities of Microorganisms", "The Control of Microorganisms", "Sources and Modes of Infection", "Infection and Immunity", "Introduction to the Study of Pathogens", "The Pathogenic Bacteria", "Pathogenic Organisms other than Bacteria", and three appendices: "Laboratory Methods", "Literature and Teaching Aids" and "Classification of Medically Important Schizomycetes".

Under the heading of "Pathogenic Organisms other than Bacteria", rickettsiæ, viruses, actinomycetes, fungi, protozoa and worms are discussed as fully as is practicable in a book "designed as a text for student nurses". A description of each organism is followed by that of the disease it causes. The lucid text and simple diagrams tracing the methods of spread of each organism indicate means of control and nursing care desirable to prevent ward infections. Care is taken to ensure that nurses should realize what specimen is required for laboratory diagnosis of each disease, and the measures necessary to ensure that it will reach the laboratory with the suspected organisms still viable.

It is unlikely that many Australian nurses would have the opportunity to practise the technical methods described, but these are interesting as an indication to nurses of the manner in which laboratory work is done. This book should be readily understandable to the layman who is interested, and most useful to all nurses, either as text or reference book.

MODERN TRENDS IN PÆDIATRICS.

"MODERN TRENDS IN PÆDIATRICS" is the title of a splendid book in the "Modern Trends Series" of Butterworth's Medical Publications.[2] It has been written by experts, most of whom are world-famous professors, and edited by Sir Leonard Parsons, the doyen among pædiatric teachers in England. In a very interesting preface, Sir Leonard Parsons expounds the statement that the "recognition of the importance of the subject is certainly the outstanding modern trend in pædiatrics in the United Kingdom". In this preface he mentions the comparatively recent establishment of numerous chairs in child health in the medical schools.

The contributions are from pædiatric professors in Sydney, Belfast, Birmingham, Leeds, London, Toronto, Sheffield, Cincinnati, Durham, Stockholm, Manchester, Amsterdam and Bristol. The special corners have been filled out by a physiologist, a pharmacologist, a pathologist, a radiologist and a thoracic surgeon. This galaxy of talent has arrayed a brilliant set of expositions of the knotty problems which are in the forefront of pædiatrics today. They have also let us have a foretaste of research projects nearing completion which are a guarantee of further triumphs to come.

It is reasonable to assume that each of the authors has taken a subject in which he has an especial interest and who has provided previously published evidence of that interest. We have then a volume which is more authoritative than would be possible if it had but one author. This is not a text-book, but the contents are admirably arranged and indexed and are very comprehensive. All doctors who take a professional interest in preventive and curative medicine and certainly those who undertake the medical care of children must read this book carefully and should be sure of the accessibility of a copy for reference.

The publishers deserve our appreciation. The book is a delight to handle as it is well bound, the type is clear and the paper and illustrations are very satisfactory.

[1] "Microbiology: With Applications to Nursing", by C. J. Witton, M.A.; 1950. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 9" × 6", pp. 710, with many illustrations.

[2] "Modern Trends" in Pædiatrics", edited by Sir Leonard Parsons, M.D., F.R.S., F.R.C.P.; 1951. London: Butterworth and Company (Publishers), Limited. 10" × 7", pp. 612, with 113 illustrations. Price: 80s.

COLLOQUIA ON ENDOCRINOLOGY.

SINCE June, 1949, the Ciba Foundation has held nine international symposia on endocrinology at which outstanding workers from many countries participated. Two of these are now published in one volume, "Colloquia on Endocrinology"—Book I, Steroid Hormones and Tumour Growth; Book II, Steroid Hormones and Enzymes. In Book I there are seventeen papers in four groups by prominent workers in cancer research on "The Induction of Normal and Malignant Growth with Steroids and Related Substances", "The Mammary Gland", "Steroids in Cancer Therapy", and "Clinical and Metabolic Effects of ACTH and Cortisone in Neoplastic Diseases". Each paper is followed by a useful discussion. Running through most of the papers is the idea that neoplastic disease may be associated with aberrations in the secretions of the endocrine glands which produce steroid hormones, in particular the adrenals and gonads, perhaps a causal relationship. The treatment of mammary and prostatic tumours with gonadal hormones is also discussed fully. The treatment of leuchæmic diseases with adrenal hormones is dealt with at length. The papers are all of a very high standard, but, of course, are for the specialist.

In Book II, "Steroid Hormones and Enzymes", there are thirteen papers with discussions. Only a few of the papers in this section are given in full, most are given as abstracts by the authors. Seven of the papers deal with β-glucuronidase and glycuronides. The rest are concerned with a variety of enzymes. These papers are all highly specialized.

The book is invaluable for the cancer research worker and the research biochemist. A medical man, wishing to know something of the latest developments in cancer research and endocrinology, would find much to interest him in the book, but the going is hard.

THE PATHOGENESIS OF TUBERCULOSIS.

IN reviewing the first edition of "The Pathogenesis of Tuberculosis" by Arnold R. Rich, the late Max Pinner described it as a book which as a whole far surpassed in interest and value any previous study in tuberculosis that he knew.[3] Many students of the subject will endorse this view. In addition to enjoying a wide popularity in English-speaking countries, this edition had the distinction of being translated into Spanish. Following a period during which it has been unobtainable it has now made a welcome reappearance in its second edition. It is likely to remain a standard work for years to come.

Although seventy-four references have been added to the already extensive bibliography, no outstanding advance in the understanding of the pathogenesis of the disease has

[1] "CIBA Foundation Colloquia on Endocrinology: Steroid Hormones and Tumour Growth and Steroid Hormones and Enzymes", edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., assisted by Margaret P. Cameron, M.A., A.B.L.S.; Volume I; 1952. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 336, with 48 illustrations. Price: 30s.

[2] "The Pathogenesis of Tuberculosis", by Arnold R. Rich, M.D.; Second Edition; 1951. Oxford: Blackwell Scientific Publications. 10" × 6½", pp. 1056, with 89 illustrations. Price: 107s. 6d.

been recorded. The use of streptomycin is mentioned and reference is made to some of the more recent work in B.C.G. vaccination.

The significance of "Much granules" is still not determined and the evidence relating to suggestions of a pathogenic filterable form of the tubercle bacillus is considered to be altogether insufficient to be regarded as convincing at present.

At the present time one hears of over-sanguine enthusiasts speaking glibly of the eradication of tuberculosis in fifteen, twenty or some other number of years: the warning against this ill-considered optimism is timely. This is in line with recent statistics relating to a number of countries having low death rates from tuberculosis, which indicate that during the years between 1940-1950 there was little correlation between the substantial fall in the death rate and the number of new cases notified, which in some countries showed an increase.

The fact that people in the more favoured countries of the world are being infected at a later age than formerly has led some authorities to the belief that progressive tuberculosis is more likely to follow a primary infection incurred in adult age. Rich believes that there is no conclusive evidence that the adult is either at an advantage or a disadvantage in coping with a primary infection as compared with a child which has developed "serological maturity".

An omission which comes to mind is the absence of a discussion on the role of the bronchus in the development of tuberculous cavities and of the fate of the blocked cavity, points of considerable practical interest to clinicians treating tuberculosis.

The bibliography includes 1500 references. A complete author index and an adequate subject index are provided. The paper is of excellent quality and there are many illustrations, mainly reproductions of pathological specimens and microscopic sections.

THE SPECIALTIES IN GENERAL PRACTICE.

R. L. CECIL's book is intended to help general practitioners, especially those who work in areas which are not readily accessible to specialists, in the solution of their everyday problems of diagnosis and treatment.[1] It is excellently printed and well illustrated, and is divided into fourteen chapters; it commences with minor surgery, and ends with psychiatry. In the last-mentioned lucid definitions and descriptions of abnormal conditions and mental states are briefly set out. A tabulated fracture guide divided into columns sets out for each fracture its appropriate first-aid, complications, anæsthesia, reduction, immobilization and convalescence. The art of eliciting a correct and complete history in all pathological conditions is emphasized. The book is full of information useful to the general practitioner and covers the entire field of medicine.

MONOGRAPHS ON SURGERY, 1952.

"MONOGRAPHS ON SURGERY, 1952"[2] is the third of a series replacing the formerly well-known "Nelson's Loose-Leaf Surgery". A beautifully bound volume superbly printed on glossy paper with a long list of contributors makes a first impression that all this must be very good. However, frankly, the book is disappointing. For example, the table of classification of renal neoplasms runs to such vague entities as:

"V. Secondary Tumours of the Kidney.

A. Involving the Kidney by Direct Extension.

(1) By extension from a retro-peritoneal node metastasis from a primary tumour elsewhere."

Two pages are devoted to a discussion of renal lipoma, a subject which seems a little unreal. On the other hand, the clinical picture of adenocarcinoma, "80% of all renal tumours", is sketchily drawn, and anyone seeking information on the important matter of the radiography of these tumours will find only a brief paragraph, with little to help him, and not a single illustration.

Whipple's article on pancreatico-duodenal cancer is a brief, but, of course, competent, review by one of the pioneers of this branch of surgery. The article on the surgery of large arteries contains much interesting information—historical, physiological and technical. Adequate accounts are given of the treatment of fractures of the femoral neck, and of the internal fixation of long bones for fracture (though with little on the indications for the use of this method).

Accounts of highly specialized topics (such as radio-active iodine in thyroid disease) represent an interesting review of new developments; in some cases, a stimulating introduction. Practising surgeons will not be encouraged by the discursive style of writing of several of the contributors.

HISTOPATHOLOGICAL TECHNIQUE.

"HISTOPATHOLOGICAL TECHNIC", by A. A. Krajian and R. B. H. Gradwohl,[1] is the second edition of a book previously called "Histological Technic" and written by Dr. Krajian alone. Its main purpose appears to be as a short reference book. The methods described are modern, and emphasis is given to the cutting and staining of frozen sections. A section (written by Dr. Lorimer Rutty) devoted to the care and use of microtome knives is particularly interesting, as the author makes a brave and competent attempt to replace what has been an occult and inexact art with a science based on careful observation. It is to be hoped that the conservatism of technicians on this subject will not prevent them from trying these new methods. The pages devoted to special staining methods are adequate for the scope of the book and appear to be soundly based on experience; discussion on the advantages and disadvantages of the various methods would have been welcome. Some sections of the book are of little use. The chapter on equipment for section cutting is one; the instructions on the use of the "Autotechnicon" are saturated with propaganda; the vague paragraphs on the recognition of cancer cells in stained smears can help no one; and the pages on the mounting of museum specimens make no mention of the use of plastic materials for containers. However, the book is worth a place on the laboratory shelf for quick reference of modern histopathological technique.

ANOTHER NEUROANATOMY.

OF the making of books on neuroanatomy there is no end—so far as the United States is concerned, at least. Such prodigality reflects both the interest this growing subject compels and the dissatisfaction of most teachers with the texts available at present. In time, no doubt, the fittest will survive. Professor Buchanan's system, in his book "Functional Neuro-Anatomy: Including an Atlas of the Brain Stem",[3] is to enlist physiology and pathology to give meaning to anatomy. This is a good approach, if not original, but it involves the risk of falling between two or more stools. This the author has not escaped, for it is evident that his physiology and pathology have been taken at second hand, and uncritically—for example, he quotes Fulton freely, but fails to distinguish toe-fanning from the Babinski response proper. He deals unevenly with his references, too, and despite a moderately up-to-date chapter on blood supply, reproduces a figure of the striate arteries (from Villiger) which is practically unchanged from that of Duret of the 1870's. Another feature of the book is insistence

[1] "The Specialties in General Practice", edited by Russell L. Cecil, M.D.; 1951. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 10½" × 7½", pp. 834, with 470 illustrations. Price: £6 17s. 9d.

[2] "Monographs on Surgery, 1952", edited by B. Noland Carter, M.D., Ph.D. Advisory editors: Gynæcology, Joe V. Meigs, M.D.; Urology, Charles Huggins, M.D.; Orthopædic Surgery, Alfred R. Shands, M.D.; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 7½", pp. 442, with 150 illustrations. Price: £7 14s. 3d.

[1] "Histopathological Technic: Including a Discussion of Botanical Microtechnic", by Aram A. Krajian, Sc.D., and R. B. H. Gradwohl, M.D.; Second Edition; 1952. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9" × 6", pp. 362, with 131 text figures and seven colour plates. Price: £3 11s.

[3] "Functional Neuro-Anatomy: Including an Atlas of the Brain Stem", by A. R. Buchanan, M.D.; Second Edition; 1951. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10½" × 7½", pp. 324, with 273 illustrations, 19 in colour. Price: 80s. 9d.

upon treatment of long fibre pathways as a whole, from end to end. This approach also has its merits, but it carries the student into unknown regions and, if the illustrations are not particularly clear—and here they are not—is likely to lead to confusion. Since each section is treated as a whole, with its own physiology, pathology and blood supply, there is a tendency for any errors to increase by geometrical progression. There is a list of references, some of which can have been consulted only cursorily, since they differ from those given in the text. The references, too, are very restricted, few being over twenty years old. Thus one misses a host of workers on fundamental aspects of the subject. Had the author's background been sounder he would not, for example, have ascribed to Mettler the discovery that the *striæ medullares* are really arcuate fibres; that was first demonstrated by Elliot Smith forty years earlier. The atlas provided is useful but not unique, and we can only conclude that this book has few, if any, advantages over any other in the same field, and the price is very high.

TESTS FOR COLOUR BLINDNESS.

THE tenth edition of Professor Ishihara's "Tests for Colour-Blindness" contains six new plates which add to the comprehensiveness of this remarkably ingenious qualitative colour vision test.[1] It is, within rather narrow limits, a quantitative test, too.

This well-known method of detecting colour blindness, popularly known as the confetti test, has never been accepted by the navy, army and air forces as reliable enough, for it does not measure the minimum thresholds of light intensities in relation to colour perception and differentiation. It remains, however, the quickest method for everyday clinical detection of red-green colour blindness. Incidentally, one observer reports that he has never, in thirty years, found a woman who could not interpret the plates correctly.

The test, which is based upon the fact that blue and yellow appear remarkably bright to red-green blind people, is a contrast pattern phenomenon; it is of interest to recall the use of red-green blind men by the air force to detect camouflaged positions.

An extraordinary feature of this otherwise excellent publication is the childishly imperfect language of the English text. There are also French and German texts. On reading the French text, it would appear that a Frenchman translated from the Japanese into the French text, which is very good, and then wrote the English text, incorporating many particles unnecessary to "the" English. One amusing sentence is this: "We have already come across with several cases of this type."

By the way, it is a pity that red and green are traffic signals. Blue-yellow blindness is practically unknown.

THE SCIENCE OF HEREDITY.

J. S. D. BACON has in his very inexpensive book, "The Science of Heredity", given both to the general reader and to the elementary science student an introduction into the recent studies of the science of genetics.[2] The language is not highly technical, though, of course, certain scientific terms belonging to the science must necessarily be used. The opening chapters give a general idea of Darwin's theory on the origin of species and the fundamental principles of heredity as discovered by Mendel, together with a simple description of the typical cell, ordinary cell division, and the reduction division which takes place in the formation of the sex cells. The following chapters are devoted to the detailed study of the nucleus of the cell with its chromosomes, which contain the genes or the bearers of the hereditary characters. The behaviour of chromosomes and the results of recent researches into the nature of genes are set out. In the final chapter Dr. Bacon reviews the impact of the genetical theory on the life of man.

[1] "Tests for Colour-Blindness", by Shinobu Ishihara, M.D., Dr.Med.Sc.; Tenth Edition; 1951. Nippon Isho Shuppan Company, Limited. London: H. K. Lewis and Company, Limited. 8½" × 6", with 38 plates in colour and "Key" in English, French and German. Price: 75s.

[2] "The Science of Heredity", by J. S. D. Bacon, M.A., Ph.D.; 1951. London: Watts and Company. 6½" × 4½", pp. 200, with many illustrations. Price: 3s. 6d.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Electrotherapy and Actinotherapy: A Textbook for Student Physiotherapists", by E. B. Clayton, M.B., B.Ch. (Cantab.); Second Edition; 1952. London: Baillière, Tindall and Cox. 9" × 5½", pp. 460, with 127 illustrations. Price: 16s.

The first edition appeared in 1949.

"Essentials of General Anæsthesia", by R. R. Macintosh, M.A., M.D., F.R.C.S., D.A., and Freda B. Bannister, M.A., M.D., D.A.; Fifth Edition; 1952. Oxford: Blackwell Scientific Publications. 9" × 6", pp. 390, with 247 illustrations. Price: 40s.

Intended for the student or for the general practitioner who wishes to refresh his knowledge.

"Œstrogens and Neoplasia", by Harold Burrows and Eric S. Horning, with contributions by W. C. J. Ross and C. W. Shoppee; 1952. Oxford: Blackwell Scientific Publications. 10" × 6½", pp. 204. Price: 30s.

An account of research into the role of œstrogens in the occurrence of neoplasia.

"A Short Practice of Surgery", by Hamilton Bailey, F.R.C.S. (England), F.A.C.S., F.I.C.S., F.R.S.E., and R. J. McNeill Love, M.S. (London), F.R.C.S. (England), F.A.C.S., F.I.C.S., with pathological illustrations by L. C. D. Hermitte, M.B., Ch.B. (Edinburgh); *Ninth Edition*; 1952. 9" × 6", pp. 1262, with 1234 illustrations, 272 in colour. Price: 55s.

All chapters have been revised or rewritten since preparation of the last edition (1948-1949).

"The Singer's and Actor's Throat: The Vocal Mechanism of the Professional Voice User and its Care in Health and Disease", by Norman A. Punt; 1952. London: William Heinemann (Medical Books), Limited. 9" × 6", pp. 100, with four text figures. Price: 10s.

A book mainly for those whose profession entails considerable use of the voice, but also for those concerned with the training of professional voice users.

"Primer on Alcoholism", by Marty Mann, with a foreword by T. Ferguson Rodger; 1952. London: Victor Gollancz, Limited. 7½" × 5", pp. 160. Price: 8s. 6d.

For alcoholics and those in contact with them.

"The Pathogenesis and Treatment of Thrombosis: With a Clinical and Laboratory Guide to Anticoagulant Therapy", by Irving S. Wright, M.D.; 1952. New York: Grune and Stratton. 9" × 6", pp. 78, with 27 illustrations. Price: $3.00.

Existing hypotheses are outlined and questions raised for future consideration.

"Circulatory Dynamics: Physiologic Studies", by Carl J. Wiggers, M.D., Sc.D., F.A.C.P.; 1952. New York: Grune and Stratton. 9" × 6", pp. 108, with 44 illustrations. Price: $4.00.

For "investigators and progressive practitioners interested in human circulatory dynamics".

"Clinical Progress in Cardiovascular Disease", edited by Herrman L. Blumgart, M.D.; 1952. New York: Grune and Stratton. 9" × 6", pp. 144, with six illustrations. Price: 4.50.

Deals with atherosclerosis, acute cardiac emergencies, surgery for mitral stenosis, cardiac patients and surgery, and emotion and the circulation.

"CIBA Foundation Colloquia on Endocrinology: Hormones, Psychology and Behaviour and Steroid Hormone Administration", edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., assisted by Margaret P. Cameron, M.A., A.B.L.S.; Volume III; 1952. London: J. and A. Churchill, Limited. 8" × 5½", pp. 396, with 78 illustrations. Price: 35s.

Contains individual papers and the discussion that followed their presentation.

"Controlled Parenthood: A Practical Handbook on Birth Control", by Reynold H. Boyd, M.B., Ch.B. (N.Z.), F.R.C.S. (Edin.); Fifth Edition; 1952. London: Research Books, Limited. William Heinemann (Medical Books), Limited. 7½" × 5", pp. 68, with 17 illustrations. Price: 5s.

The title is self-explanatory. The book is meant for the intelligent "lay" public.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

A MESSAGE TO MORTALS.

ONE of the peripatetic correspondents of *The Lancet* last year[1] lamented in verse his inability to understand a symposium by a group of social psychologists. He concluded with an appeal:

> Dear Social Psychologists, in conversation
> The Gods on Olympus use what terms they may.
> But a Message to Mortals requires a translation,
> Or they're apt to believe you have nothing to say.

It is unlikely that the psychologists will take this too hardly, as most of them will admit that some at least of their number can on occasion enshroud a comparatively straightforward idea in such jargon that it is hidden from the eyes of ordinary mortals. It is true, of course, that some of the ideas they seek to convey cannot be readily put into words, and a new vocabulary becomes unavoidable. Moreover, it is hard to translate the language of Olympus, and the psychologist ends by being "not understood" in more than one sense. The psychiatrist to a large extent shares with the psychologist these disadvantages of an Olympian outlook, but probably suffers more from greatness thrust upon him. The nearly magic powers attributed to him by certain modern films and popular periodicals are an embarrassment, in which he has all our sympathy; but unfortunately in this respect his foes can be of his own household. A striking example of this is seen in the president's address last year to the Section of Psychiatry of the Royal Society of Medicine, in which Desmond Curran[2] takes to task Dr. Brock Chisholm, Director-General of the World Health Organization, for "expansionist claims" in relation to psychiatry. Curran first of all falls out with Brock Chisholm's "warm approval" of the World Health Organization's definition of health: "Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity." This, Curran states, seems logically to imply that everybody—in this world—is sick; and illness is equated in the average mind with irresponsibility. However, Brock Chisholm has gone further and advocated that the chief concern of psychiatry should be with preventive measures and "positive health", that the technical guidance needed by parents, educationalists, politicians and many others can come only from psychiatrists, and that in psychiatry lies the best hope of resolving international tensions and so preventing war. Obviously embarrassed by all this and opposed to much of it (he classes as "grandiose" the claims about resolving international tensions), Curran descends from the psychiatric Olympus and with apparent relish sets himself to show that he and his fellow psychiatrists are mortals as other men are. He considers the "dangers of expansionism" in four connexions: crime and criminal responsibility, working capacity, selection and psychotherapy. Speaking of crime and criminal responsibility, he points out that in general medical claims appear to be three: that the sick should be regarded as medical responsibilities, that sickness implies irresponsibility or diminished responsibility, and that the criterion of sickness is the demonstration of a pathological condition. If, however, an epileptic commits a crime (say, murder) that shows a capacity for rapid adaptation to altering circumstances and other features not consistent with epileptic confusion or automatism, is the operation of the law to be modified if he has an abnormal electroencephalogram? If so, preventive measures should be taken, and logically those with abnormal electroencephalograms might be deprived of the right to enter into business contracts and other civil rights. This would be intolerable, but the alternative is to make the abnormal electroencephalogram a criminal asset. As a further development, it is obvious that all criminals lack complete social well-being, and so by the World Health Organization definition of health they are all sick. Crime thus becomes a job entirely for the doctor. Before long we may find all criminals being regarded as sick men and *ipso facto* irresponsible—another intolerable situation. Yet, as Curran remarks, "it is to be feared that the comparable psychiatric evidence is too commonly given in the courts". Turning to the question of working capacity, Curran points out that as the doctrine of positive health spreads, inefficiency tends to become a medical problem, and an impossible burden falls on psychiatry. Indeed, the tendency already exists to refer the inefficient, the ineffective and even the "work-shy" for medical rather than executive disposal (an old bone of contention only too familiar to the medical officer in any of the armed services). Curran insists soundly that any opinion expressed in these circumstances should be related to the medical findings, positive or negative, and that the doctor should be wary of entering into social and other aspects that are not part of his job. On the subject of selection (for example, officer selection in the services) Curran warns psychiatrists of the dangers of venturing into "positive selection". The traditional medical role in selection, he points out, has been one of exclusion, and that is difficult enough. If a doctor can contribute something worth while to the positive aspects of selection, it is more likely to be due to personal knowledge or judgement than the result of his psychiatric training. In addition, participation in cooperative positive selection procedures tends to lead to breach of professional confidence in one way or another. On these grounds Curran recommends that when a report is given to lay people on selection matters, it should be

[1] *The Lancet*, October 20, 1951.
[2] *Proceedings of the Royal Society of Medicine*, March, 1952.

confined to the statement that the doctor can or cannot find medical reasons against acceptance. As the last of his four subjects affected by "expansionist" claims Curran deals with psychotherapy (by which he appears to mean more particularly analytical psychotherapy) and the unjustified results that for one reason or another have come to be expected from it. He does not decry its real value for one moment, but insists that its application is practicable in only a limited number of cases. Happily, he remarks, many patients get better without analysis, and for many it is best to get them to accept their limitations.

On the positive side Curran advocates greater attention to clinical studies in psychiatry (he seems a little tired of the current enthusiasm for "dynamic" concepts) and makes practical suggestions for the training of medical students and nurses, pointing out that "the majority of psychiatric patients must necessarily be tackled by those who are not psychiatrists". In conclusion, he draws attention to the vast fields of psychiatry's lawful domain and to all that remains to be done; he asks if it is necessary or wise and if it is not premature "to look for fresh fields to conquer or to fail in". It must not be thought that he deprecates his own specialty; on the contrary, he is very loyal to its best interests. However, he prefers to think of it as "Psychiatry Ltd.", a respectable limited liability company with responsible shareholders, who "are vexed when others undermine the reputation of the firm by using the name to float bogus companies, with grandiose prospectuses, backed up by balance sheets that do not add up to make sense". These are provocative words and are not likely to go unchallenged. No doubt also Curran overstates his case in places, but his views, carrying as they do the weight of a president's address to a section of an important medical body, are a healthy corrective to certain extreme tendencies in psychiatry, tendencies that have done no good to a specialty that has been toiling slowly but effectively to establish itself in the confidence of the main body of the medical profession and of the community in general.

Current Comment.

PRELIMINARY REPORT ON A NEW ANTIBIOTIC.

EARLIER in the present year a description of a new antibiotic substance was published by J. M. McGuire and a number of associates. This has been named ilotycin or erythromycin, and is derived from *Streptomyces erythreus*; its general properties coincide roughly with those of penicillin, but it has in addition some other interesting features. Another study of some of its bacteriological and clinical features has just been published by Fordyce R. Heilman, Wallace E. Herrell, William E. Wellman and Joseph E. Geraci.[1] These writers refer to the substance as erythromycin. It is basic in nature, and goes into solution slowly, but can be more readily dissolved after initial grinding with a little alcohol. Solutions of one to two milligrammes per millilitre have been found to remain stable for several weeks when frozen, but it remains active in physiological saline at room temperature for only twenty-four hours, and loses activity slightly after three days under similar conditions. Sensitivity tests have been carried out under controlled conditions. These show that the Gram-positive organisms are more sensitive than the Gram-negative. Fungi were resistant, so, too, were the enteric group of bacteria, but the *Hæmophilus pertussis* was quite sensitive. An interesting feature was that the Corynebacteria were among the most sensitive organisms tested; this seems to have considerable potential importance, since the *Corynebacterium diphtheriæ* was one of the most highly sensitive, as judged by the results of tests on fourteen strains. Group A hæmolytic streptococci and *Diplococcus pneumoniæ* were also among the very sensitive organisms tested. One item of interest mentioned among these bacteriological tests is that the authors found that old degenerate strains of Corynebacteria which had been subcultured a long time, and had assumed a coccoid form, were found to have developed considerable resistance. Such strains were not included in the lists prepared of bacteria found to be sensitive. Tests of the bactericidal properties showed that erythromycin had a variable but not rapid action; certain organisms were not killed at all, while penicillin was under similar conditions quite active. Observations on cross-resistance to organisms which had acquired or possessed resistance to other antibiotics did not suggest that erythromycin was in this respect inactive. Further investigations showed that the substance reduced the numbers of Gram-positive bacteria in the fæces when given by mouth, and other tests established that it was diffused into pleural and ascitic exudates, and also into the cerebro-spinal fluid and the bile. It is apparently concentrated in the liver. Large amounts of erythromycin were proved to be excreted in the urine. Finally, serum concentrations were investigated; these indicated that the ideal spacing of doses was six hours.

Clinical trials were carried out on the basis of these investigations; the usual method of administration was by mouth, and the recommended dose for the average adult was 0·4 to 0·5 gramme every six hours. Doses exceeding this amount tended to produce gastro-intestinal irritation, but doses lower than 0·3 gramme failed to secure an adequate serum level. These trials suggest that erythromycin can be used for the same types of infection as penicillin, but the authors suggest that a promising field for its use appears to be the group Corynebacterium, against which it has a high degree of activity. Another special field may be in infections by *Micrococcus pyogenes*, as erythromycin appears to be effective against strains of this organism which are resistant to other antibiotics. These trials are, of course, only preliminary, though a considerable volume of work has been carried out, and in view of the limitations of certain types of antibiotics in the light of extended experience, the writers are careful to state that more clinical studies must be made before the new antibiotic can be assessed for its true value.

[1] *Proceedings of the Staff Meetings of the Mayo Clinic*, July 16, 1952.

SIR WILLIAM OSLER AND HIS LEGEND.

A DISTINGUISHED British surgeon of the present century expressed his opinion that "from the moment of a man's death the memory of him begins to fade and that nothing can arrest this decline; for in our commemorations we make our own ideals and on them we drift away from the reality of him whom we would commemorate". These words are found in the introduction to a small book, "Osler: The Man and the Legend", by W. R. Bett,[1] who seeks to explain the unusual happening of a name becoming almost legendary soon after his passing, and to give more substance to that legend while the subject of it is still fresh in our minds. Many of the earlier chapters are taken up with some interesting reflections upon the great physician's attitude to important diseases that were prevalent in his day: typhoid fever, pulmonary tuberculosis, the pneumonias, syphilis, aneurysm and diseases of the heart. But there is more pleasurable reading in the later chapters touching on the various spheres of learning and culture which Osler was able to interpret so vividly

[1] "Osler: The Man and the Legend", by W. R. Bett; 1951. London: William Heinemann (Medical Books), Limited. 7¼" × 5", pp. 132, with six illustrations. Price: 15s.

to his students and professional brethren. Few will disagree with Bett's summing up of the reasons for the premature development of an Oslerian legend in these words:

> Many of the qualities for which Osler is remembered and revered were attributable to his love of the classics—his philosophy, breadth of vision, serenity, courage, humility, power of expression, and humanity all told of a man who had more than an academic interest in the writings of the great men in ancient times, and like those ancient Greeks he dwelled essentially in the sunshine of life.

Nevertheless, one cannot resist the feeling that the progress of this legendary figure in its ascent to the Olympian heights might be greatly accelerated if publishers could be persuaded to give more thought to the dissemination of Osler's own writings and rather less to the production of books written about the great man.

THE ARTIFICIAL KIDNEY.

THE Medical Clinic of the University of Lund, Sweden, has, under the direction of Nils Alwall and with the help of his associates, carried out a long series of researches on the improvement, the physical principles and the clinical use of artificial dialysing methods in ridding the blood of accumulated poison threatening death. A recent number of *Acta medica Scandinavica* contains four articles[1] entitled "On the Artificial Kidney", which embody the results of what appears to be very careful work in both laboratory and hospital. The articles deal respectively with the efficacy of the modified dialyser, suitable equipment of localities for the dialyser, treatment of severe phenobarbital poisoning in rabbits together with a preliminary report on dialytic treatment of barbiturate poisoning in patients, and finally the efficacy of the dialyser-ultrafilter intended for human use. It will be remembered that in the artificial kidney a thin film of heparinized blood moves through the force of the left ventricle between "Cellophane" walls, which separate it from the dialysing fluid, usually a Locke solution. The articles make it clear that where a critical condition has arisen, such as, to quote the quaint English of the final contribution, "life threatening lung œdema" or "life threatening cerebral œdema", the artificial kidney can restore the blood to something close to normality. The main criticism which can be advanced against this rather heroic procedure is that where the deleterious matter is being constantly added to the blood, as in uræmia, the artificial kidney is purely palliative and merely postpones the ultimate catastrophe. In cases of extraneous poisoning, as from overdose of a drug or from accidental or suicidal administration of a lethal chemical, the artificial kidney can be acclaimed as giving hope for a cure unless the injury to the living machinery of the body has gone too far.

MORTIMER'S MALADY.

THE first description, given in 1875, of the disease entity best known in Australia as Boeck's sarcoid, sarcoidosis or Besnier-Boeck-Schaumann's disease was the product of Sir Jonathan Hutchinson's remarkable powers of clinical observation. Twenty-three years later, nine years after Besnier had given the name *"lupus pernio"* to a most striking form of the disease, Hutchinson described two cases under the name "Mortimer's malady", after a woman sufferer. Apparently neither the gallantry nor the justice of this gesture appealed to subsequent writers, for, by virtue of one of the vagaries of eponymic nomenclature, the names of Mortimer and Hutchinson are not usually associated with the disease. Boeck, in a series of papers commencing in 1899, recognized that internal viscera, mucous membranes, lymph glands and bones might be affected as well as the skin. It is interesting to note that an autopsy was performed on the patient in Boeck's first case in the year 1940, no evidence of the disease being found to remain. In a comprehensive monograph written in 1914, J. Schaumann conclusively established the identity of lupus and Boeck's sarcoid of the skin, and he presented the first autopsy findings. Pointing out that the tuberculin reaction was usually of the negative type, he considered the disease to be a form of infectious granuloma unrelated to tuberculosis in spite of the similar histological features in some respects.

These facts are related by W. T. Longcope and D. G. Freiman[1] in a thoughtful monograph of 120 pages devoted chiefly to the natural history and the clinical and pathological features of the disease. On the vexed question of the relationship of sarcoidosis to tuberculosis, they suggest that the histology of the lesions and their distribution present significant differences, and that such bacteriological and serological investigations as indicate their identity are at best inconclusive. Mention is made of the failure of B. Robinson and A. Pound[2] to isolate organisms by culture or animal inoculation, a finding reported in this journal two years ago. The Kveim test, based on the production of a delayed and prolonged local reaction to the intracutaneous injection of an emulsion prepared from a proven skin lesion, has so far given negative results in cases of tuberculosis and positive ones in most cases of sarcoidosis, but nevertheless its precise significance is not yet clear. The possible, but unlikely, relationship of leprosy, brucellosis, histoplasmosis and certain chemical substances (notably beryllium) to sarcoidosis is discussed. As a rule, a diagnosis should not be based on the histological appearances alone, particularly if other causes of similar granulomatous lesions have not been excluded.

The disease appears commoner amongst Negroes and in rural districts. Its onset is usually insidious and a number of symptomless cases are found on routine radiological examination. The common presenting symptoms are loss of weight, malaise, cough, shortness of breath, ocular disturbances, skin eruptions and lymphadenopathy, but many others are observed. Longcope and Freiman point out that a precise clinical account applicable in general is impossible in a disease which "pursues such an erratic and unpredictable course, and is subject to such unexpected recoveries, and remissions with subsequent unaccountable relapses", so that they adopt a regional classification to discuss the various manifestations at some length. An interesting observation selected from a wealth of information is that pain is relatively rare, probably because the lesions do not usually involve nerve roots, serous surfaces or periosteum; even in eye affections the pain seems less intense than in other diseases. Hyperglobulinæmia is perhaps the most constant biochemical abnormality. Hypercalcæmia may occur independently and is not necessarily indicative of bone involvement, which may be found in the presence of normal serum levels of calcium, phosphorus and phosphatase. Brief consideration of the course, prognosis and treatment occupy the remaining few pages of Longcope and Freiman's paper. Favourable response to cortisone and ACTH has been recorded, and, if confirmed, would favour the non-tuberculous origin of sarcoidosis.

It remains to note two further papers on this disease by W. E. Jacques,[3] the first a case report and the second a review of the literature with some additional observations. He describes a case which he attributes to hypersensitivity to *Strongyloides stercoralis* and another in which foci of necrotizing arteritis were found. Adducing further evidence from a consideration of the relevant literature of sarcoidosis and nematode infestation, he suggests that the latter may be the source of antigens to which, in some instances, the host reacts abnormally to produce the lesions of sarcoidosis. The evidence presented is worthy of study, but is not, on the whole, convincing. Longcope and Freiman justly observe that "the most telling contribution that could be made to our knowledge of this peculiar condition would be the discovery of its ætiology". The present situation reminds one of the discussions on the ætiology of pellagra of a generation or so ago.

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[1] *Acta medica Scandinavica*, Volume CXLIII, Fasciculus IV, 1952, pages 288, 296, 299 and 307.

[1] *Medicine*, February, 1952.

[2] THE MEDICAL JOURNAL OF AUSTRALIA, April, 1950.

[3] *Archives of Pathology*, June, 1952.

Abstracts from Medical Literature.

SURGERY.

Congenital Atresia of the Sigmoid Colon.

MARK E. GANN AND ELMER HOFFMAN (*The American Journal of Surgery*, April, 1952) state that the outlook in cases of congenital obstruction of the intestine is improved as a result of the following factors: earlier diagnosis, improved pre-operative and post-operative care, use of antibiotic agents, blood, plasma, parenteral fluid therapy and vitamins, better anæsthesia, improved suture material, and better operative techniques. Even with all of these life-saving features a complete review of the literature on the subject is said to reveal only two previously reported cases of successfully treated congenital atresia of the colon. The authors report a third case. They state that atresia of the intestine may assume one of two forms, namely, that of a blind ending of the intestine giving discontinuity (this may be multiple) and that of a completely blocking intestinal diaphragm. In their case the latter was found. Restoration of continuity was achieved by a two-layered, side-to-side anastomosis between parts of the sigmoid colon proximal to and distal to the area of the atresia. During the operation 50 millilitres of blood were given intravenously (the child then weighed eight pounds five ounces). The post-operative regimen included use of an oxygen tent, naso-gastric suction, rectal intubation, intravenous fluid therapy, and administration of vitamins. Injections of liver extracts, penicillin and streptomycin were also given. Apart from mild alkalotic tetany, which responded to the intravenous injection of calcium gluconate and to the intramuscular injection of 32 milligrammes of sodium pentobarbital, the post-operative course was practically uneventful.

Dehiscence of Abdominal Wounds.

M. G. BAGGOT (*Current Researches in Anæsthesia and Analgesia*, September-October, 1951) discusses the cause of dehiscence of the abdominal wound. He suggests that during laparotomy, air enters and forms pockets, virtually an acute pneumoperitoneum. He has conceived the idea that burst abdomen is to a great extent caused by the intraperitoneal trapping of a large volume of air when the abdomen is closed post-operatively. He also states that if the abdomen is still distended the surgical closure should wait, tight strapping being used to protect the wound till the abdominal contents have returned to their usual capacity.

Oleogranuloma of the Rectum.

DAVID A. SUSNOW (*The American Journal of Surgery*, April, 1952) reports six cases of rectal oleogranuloma which followed the rectal instillation of petrolatum. These instillations had been made at the end of operations in order to ease the first bowel movement, but the rectal masses did not appear for some weeks. One patient complained of a feeling of pressure in the rectum, and this was relieved by excision of the protruding mass. The author states that the diagnosis of oleogranuloma of the rectum should be easily made from the history, examination and biopsy. As there is considerable doubt whether these oily instillations really facilitate the action of the bowels, he recommends that their use be ceased. In addition, he draws attention to the granulomatous tumours which are produced by the parenteral injection of sclerosing chemicals in many of the oily vehicles such as are used for the treatment of hæmorrhoids. The granuloma is then due not to the phenol and other chemicals, but to the oily excipients. Since olive oil is an exception and produces no special tumour formation, its use is advised as the excipient in these injections.

Acute Appendicitis in the Aged.

L. CARP AND J. A. ARMINIO (*The American Journal of Surgery*, June, 1952) report the clinical and operative findings, diagnostic problems, pathology and results of treatment in 21 chronically ill patients over the age of sixty years who developed acute appendicitis. Most of the patients were substandard surgical "risks" and had multiple degenerative diseases frequently complicated by malnutrition and personality changes. In 19 cases the diagnosis was difficult. As a rule the onset was insidious. In some cases there were vague and mild abdominal cramps or discomfort, which the patient disregarded and which sometimes continued for days. The average time from the beginning of symptoms to operation was three and a half days; the minimum time was twelve hours and the maximum one week. There was usually nausea but no vomiting. Constitutional disturbance was not great. In six cases the temperature was normal. Constipation was the rule; the tongue was dry and coated and the breath foul. Three signs were almost constant: tenderness over McBurney's point, rebound tenderness, and pronounced abdominal distension simulating intestinal obstruction. A flat abdominal X-ray film showed distended intestinal loops and served to rule out renal and gall-bladder calculi. From a study of the appendices removed in the cases discussed and at autopsy from elderly subjects who had not appeared to be suffering from appendicitis, the authors concluded that the severity of the pathological processes in their series was due, firstly, to the comparative lack of lymphoid follicles in the wall of the appendix so that there was diminished local resistance to infection, secondly, to arteriosclerosis of the blood vessels which supply the appendix so that ischæmia makes it more vulnerable to gangrene, and thirdly, to the presence of fæcoliths, foreign bodies or fibrous strictures resulting from previous inflammation, all of which produce obstruction in the appendix. It is stated that additional contributing factors are a long time interval before operative therapy, catharsis, accompanying degenerative disease, and disturbance in normal chemical balance. The mortality rate in these 21 cases was 23%. The following methods of reducing the mortality are suggested: (i) early diagnosis through better education of the public and the physician; (ii) quick operative intervention after prompt supportive therapy, with appendicectomy as a routine, unless technical difficulties or the danger of spread of infection from added trauma would add to the threat of mortality; (iii) drainage in cases of peritonitis or abscess; (iv) use of the McBurney's incision, unless an exploratory laparotomy is indicated when diagnosis is doubtful; (v) non-closure of the abdominal wall above the peritoneum or fascia if gross contamination of the wound has occurred; (vi) a carefully chosen and expertly administered anæsthetic; (vii) use of the newer supportive and antibiotic therapy; (viii) early ambulation whenever possible.

Oral Carcinoma.

W. W. CARROLL (*Archives of Surgery*, May, 1952) reports 186 cases of oral carcinoma and their follow-up. He states that with oral carcinoma there is a high percentage of early lymph node metastases, which are located anatomically in such a way that primary and secondary growths can be removed in continuity. Analysis shows that such excision of glands of the neck with hemimandibulectomy and partial glossectomy has little morbidity, and the results are encouraging.

Ligation of the Inferior Vena Cava for Suppurative Pelvic Thrombophlebitis.

C. G. COLLINS *et alii* (*Surgery*, April, 1952) have analysed their results in 70 cases in which ligation of the inferior *vena cava* and ovarian vessels was performed for suppurative pelvic thrombophlebitis. They state that such ligation, whether performed by the trans-abdominal or the extraperitoneal route, is a relatively simple procedure and is not accompanied by any degree of shock. It was performed only if the patients failed to respond to medical therapy, or showed evidence of pulmonary infarction and had the worst type of pelvic infection one can encounter. Of the 70 seriously ill patients, 62 survived and eight died. Four of the eight patients who died were operated on from nine to twenty-one days after admission to hospital. Death did not result from the operation, but from delay in operating. After ligation of the inferior *vena cava* the vertebral, lumbar, azygos and portal systems are the most important collateral routes; the hæmorrhoidal plexus and the superficial veins of the trunk are the least important. Varicosities of the abdominal wall, flank, thigh or leg have not developed in any of the patients with the exception of one who had varicosities of the leg before operation. In the latter case the varicosities were not made worse by this operation. In eight cases in which laparotomy was subsequently performed, the inferior epigastric vessels were found to be of normal calibre and there was no increase in size of the pre-peritoneal vessels. Hæmorrhoids have not developed in any case. The immediate reaction of the lower extremities to ligation of the inferior *vena cava* is variable. Forty-eight patients have had no œdema at any time. Eight patients noticed occasional mild swelling of the feet after they had been up and about for many hours. However, œdema of the ankle and leg was not noticed. Six patients may be said to have had true œdema in that some swelling of the ankle and lower half of the leg to about the calf was noted except when they were at complete bed rest. None of these patients was incapacitated in any manner. Retrograde thrombi occasionally developed, but responded rapidly to lumbar sympathetic block. In nearly

all cases one or both lumbar sympathetic chains were severed at the time of ligation, or lumbar sympathetic blocks were performed post-operatively on the uninterrupted side or sides.

Therapeutic Ligation of Hepatic Artery.

H. I. CHENOWETH (*Annals of Surgery*, June, 1952) advises ligation of the hepatic artery for advanced cirrhosis of the liver producing ascites or repeated bouts of hæmorrhage from œsophageal varices. He considers that factors contributing to portal hypertension are (*a*) direct communication of arterial pressure to portal vessels through dilated capillaries, and (*b*) the larger volume flow of the hepatic artery in proportion to portal flow in cases of cirrhosis, as compared to that in normal liver. He reports two cases, but issues the warning that ligation of the hepatic artery is not an entirely innocuous procedure.

Anal Incontinence.

K. L. PICKRELL *et alii* (*Annals of Surgery*, June, 1952) report four cases of anal incontinence in children, of neurogenic origin, successfully treated by construction of a rectal sphincter with use of the gracilis muscle. The authors transplant the muscle through a subcutaneous tunnel to the perineum, retaining nerve and blood supply. They find that the patients, although lacking a sensory arc of the defæcation reflex, experience a sense of fulness in the lower part of the abdomen, and continence is maintained by voluntary contraction of the new gracilis sphincter.

Radical Ilio-Inguinal Lymphatic Excision.

A. P. HOVNANIAN (*Annals of Surgery*, April, 1952) discusses the anatomy of the ilio-inguinal region, and lists the following indications for radical ilio-inguinal lymphadenectomy: (i) cancer of the skin below the umbilical line, the perineum, the anus below the dentate line, the urethra, the scrotum and penis in the male, the clitoris, labia and lower part of the vagina in the female; (ii) selected cases of uterine cancer; (iii) primary malignant lymphoma involving these lymphatic glands. He states that because the lymphatics of the mid-line structures decussate, a bilateral dissection is imperative for malignant disease, especially for melanomata involving the umbilicus, anus, perineum and median raphe of the scrotum. The author prefers to operate on one side at a time. He recommends an incision starting at the level of and three centimetres in front of the highest point of the iliac crest. It is continued to a point three centimetres in front of the anterior superior iliac spine. Here the inguinal fold is crossed downwards and the incision is carried to the *fossa ovalis*. From there on the incision courses downwards to the level of the medial crease of the thigh. An elliptical island of skin is left on the specimen if there is a suspicion of involvement of the skin lymphatics. The dissection starts in the lower quadrant of the abdomen. The second field of dissection is in the anterior and upper two-thirds of the thigh; and the third is in the retroperitoneal pelvic lymphatic bed. The retroperitoneal space is entered by cutting the abdominal muscles 1·5 centimetres along the iliac crest parallel to the skin incision. Osteotomy is performed on the anterior superior iliac spine, and Poupart's ligament is freed from its attachment. At the end of the operation the anterior superior iliac spine is wired back in place.

Replacement of Œsophagus by Plastic Tubes.

EDGAR F. BERMAN (*Annals of Surgery*, March, 1952) reports the results of replacement of resected portions of the œsophagus in 20 dogs by plastic tubes 55 to 90 millimetres in length and 14 to 18 millimetres in internal diameter. In only one dog was there leakage at the anastomosis. At necropsy it was found that all the dogs had developed a fibrous sheath around the plastic tube, lined by epithelium, which was complete after a year. The author carried out these experiments to determine whether such a replacement would be leak-proof and whether the embedded tube would eventually cause undesirable effects. He states that the method commonly used clinically after resections of portions of the œsophagus is œsophago-gastrostomy, but that is formidable and time-consuming and invites post-operative pulmonary and other complications. The tubes used in the present operations were made of methacrylate or polyethylene plastic, which are non-porous and easily sterilized, and do not absorb water. They are non-absorbable and remain unchanged in the tissues. There is no foreign body or inflammatory reaction. Mucus does not tend to adhere to them; blood and lymph clot slowly on them.

Chylous Peritonitis.

M. THOMPSON AND W. BUSCHEMEYER (*Annals of Surgery*, May, 1952) define chylous peritonitis as a condition which is likely to be mistaken for other inflammatory conditions of the peritoneum and which is characterized by presence of chyle in the peritoneal cavity. It is distnguished from chylous ascites or chylo-peritoneum, which is a chronic, slowly developing process without sufficient irritation of the peritoneum to lead to rigidity of the abdominal muscles, fever, leucocytosis and pronounced tenderness. Many of these slowly developing cases have been due to lymphoma, carcinoma, suppurative cellulitis in the mesenteric and retroperitoneal lymph nodes, filariasis, trauma (sometimes mild and sustained days or even weeks prior to the recognition of the ascites) and venous thrombosis. Except for possible trauma, acute cases requiring an emergency operation have no obvious cause. In the authors' case an enlarged heart was present, and whether this could have been an underlying cause from pressure on the thoracic duct is not certain. However, recovery followed drainage of the lesser sac.

The Hindquarter Operation.

SIR G. GORDON-TAYLOR AND R. MUNRO (*The British Journal of Surgery*, May, 1952) present their results in 58 cases requiring the hindquarter operation. They state that from this unparalleled series they have reaped a bountiful harvest of experience in operative technique and in pre-operative and post-operative management. The pre-operative preparation includes instruction in breathing exercises, and penicillin and streptomycin therapy is started the day before operation. The details of operative technique are given at length, and it is pointed out that the chief hazard encountered is hæmorrhage from the iliac vessels and their immediate branches during the division of the posterior segment of the pelvic girdle. This danger is best avoided by observing the following points: (i) elective ligation of the common iliac vessels, (ii) division of the *symphysis pubis* before embarking on the posterior section of the girdle, (iii) use of a Gigli saw and avoidance of the chisel. It has been found that common iliac ligation has provided more certain hæmostasis when the gluteal flap is cut, and has not resulted in ischæmia of the pelvic viscera or in any significant impairment of vitality of the posterior skin flap. In cases in which the posterior flap has sloughed subsequent to the operation, the determining factor has been rather that the flap has had to be cut thin on account of posterior extension of the growth on the *dorsum ilii*, or because the tumour has already been unvailingly irradiated. The average post-operative period in hospital has been fifty-nine days. The operation has not been exempt from post-operative complications. Some degree of shock is usual, but with unilateral spinal anæsthesia, adequate transfusion and improvements in operative technique it is no longer a fearsome feature. Other complications have included primary and reactionary hæmorrhage, retention of urine (which occurs in all cases for the first forty-eight hours, but in some is more obstinate), œdema of the penis and urinary fistula.

Urinary-Vaginal Fistulæ.

J. R. LONGLEY AND L. F. GREENE (*Surgery, Gynecology and Obstetrics*, June, 1952) discuss the methods of diagnosis of urinary-vaginal fistulæ. They ask first if the fluid from the fistula is urine. If there is doubt, they inject indigo-carmine and conclude that if a vaginal pack is stained green a fistula is present. They state that the clinical features vary according to the level of the urinary tract from which the fistula arises. If it is from the ureter the discharge is constant, but the bladder seems to fill and empty normally. In post-operative fistulæ there is frequently a history of flank pains and rigors preceding the discharge. If it is from the bladder and part of the urethra, the vaginal discharge is constant and there is minimal or no voluntary micturition. The picture may be complicated by development of cystitis. If it is from the urethra, the discharge is intermittent and synchronous with micturition. The authors state that with vesico-vaginal fistula, excretion pyelography shows no abnormality unless there is involvement of the ureteral orifice. With uretero-vaginal fistula, the examination tends to show hydronephrosis and hydroureter on the damaged side. With vesico-vaginal fistulæ, cystoscopy shows cystitis and perhaps a fistulous opening. If the latter is found, the authors advise the passage of a ureteral catheter through the fistula and the insertion of a silk guide through the fistula to facilitate later surgery. With uretero-vaginal fistulæ, cystoscopy shows little or no excretion on the affected side. Vaginal examination may show the fistula; if so, a catheter should be inserted from the vagina before cystoscopy. The authors briefly discuss the principles of surgical repair.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XLI.

SQUINT, OR STRABISMUS OR HETEROTROPIA.

THERE is one thing everybody should know about squint: valuable sight may be lost if treatment is not early and thorough. Too often parents' minds are eased by the neighbour's encouragement that: "Our Tommy was cross-eyed and grew out of it." And so the cosmetic defect is neglected in the hope that it will go—a vain hope, for if it goes, so probably may the sight of one eye. The obvious or visible defect can be put right at any stage by operation if necessary, but as a rule the visual defect must be cured in the first five years of life. Frequently in later life the young hopeful is turned back from the military or public services because "there's sight only in one eye". Occasionally that one good eye suffers loss from trauma or disease, and the victim may become an object of pity, a family burden and an expense to the State.

Sometimes either eye of a child will squint alternately, but more frequently the defect is monocular. In the former case the vision is usually equal in each eye. In the latter the sight of the turned eye may not develop. It is strange that the left eye converges much more frequently than the right one. In a recent series the proportion was right 37, and left 63. Is this due to the prevailing mastery of the right eye in two-thirds of people?

Amongst 9156 private patients there were 504 blind in one eye—that is 5·5%—and amblyopia was the cause in 34 or 7·6%.

How, then, can this be prevented? Strangely enough, by the simplest possible treatment. First of all, let us consider what causes squint and the associated loss of vision.

Some children, of course, have a congenital defect of the eye or its cerebral pathway that makes the visual loss incurable. Such a defect may be due to maternal ill-health or to a difficult labour. One is struck by the frequent story of prolonged labour, and of faulty presentation or of bruising and inability to open one eye, and the presence of tell-tale scars close to the lids, suggestive of a struggle; 8% of patients in a recent series showed evidence of some birth injury. What influence on immature tissues or on unstable mechanism can a temporary anoxæmia have whilst the amniotic environment is giving place to an atmospheric one? Let us recall that inheritance may permit the passing on of some muscular, ocular or cerebral imperfection that leads to a squint during the early or later attempts at binocular fixation. Inheritance indeed appears as the most important factor in the ætiology of squint.

Squint may be convergent, divergent or vertical, but as the first-named is most common these notes will refer mainly to it. Approximately half of the subjects with convergent squint have a vertical defect as well, which sometimes makes the treatment more difficult (Figure I).

Types and Causes.

There are three types of squint: (i) the paretic type, (ii) the accommodative type, (iii) type with defective fusion.

Paretic Type.

The paretic type, unlike the others, arises just as frequently in later life as in childhood, for it may be due to trauma or to nerve diseases. Again, unlike the other types, a paretic squint becomes more pronounced in one direction of gaze than in the others, for, as this type is due to a weakness of one or more of the extraocular muscles, the squint will be most pronounced when the eyes are called on to look in the direction in which that muscle (or those muscles) normally acts (Figure II). The squint is said to be incomitant to distinguish it from the usual type of squint in children, in which there is a large degree of comitance, or very little variation in the angle of squint in different parts of the motor field. Very similar to the squints due to paresis are those, in childhood, which are due to a structural abnormality rather than to an actual weakness of one or more muscles. For example, there may be fibrosis of a muscle, an abnormal insertion of its tendon or a defect in the check ligaments that help its action through their attachment to the globe and its adjacent tissues.

The paretic origin of a squint may become obscured because of secondary changes in the other muscles. This is in accordance with Hering's law, which states that "every voluntary impulse for ocular movements arising in the frontal cortex goes equally to both eyes, to the muscles concerned in the movement". The antagonist muscle may become shortened, the synergist of the other eye may overact and its antagonist may underact. This lessens the incomitance and the squint resembles the non-paretic or concomitant type so common in early life. Difficulty in recognizing the cause may be due to paresis of a vertical rotator of the globe interfering with fusion and causing a latent tendency to a horizontal deviation to appear. This may take charge and the incomitance be difficult to find.

It is uncertain how many squints in children are due to ocular paresis. The onset of squint during or after an attack of measles or other disease of early life makes one

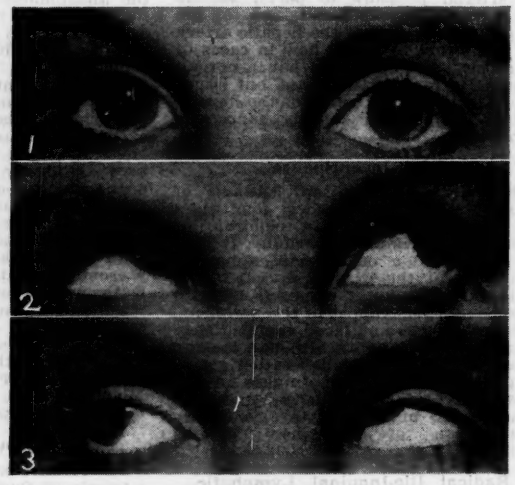

FIGURE I.

A slight convergence in central gaze (1) associated with a vertical error in looking to either side (2 and 3).

suspicious, but then why should a muscle be paralysed in such diseases except perhaps in whooping-cough or poliomyelitis? It is more likely that a cerebral centre or a supranuclear mechanism of control is upset, which in early life may be unstable and incompletely developed. The defect then will not be of the paretic type, for isolated muscles cannot be affected by an upper lesion but only by one affecting muscle, nerve or nucleus.

Accommodative Type.

Most children with convergent eyes are long-sighted. The wearing of glasses correcting the hypermetropia usually lessens and may remove the squint. Long-sightedness calls for excessive accommodation if vision is to be clear. As accommodation and convergence are synergic, it is not surprising that over-accommodation leads to over-convergence. Squints of this type may require no other treatment but the wearing of glasses. If, owing to defective sight, the use of glasses is necessary apart from the squint, no further treatment may be necessary. But if glasses are not essential for sight, orthoptics may enable these children to maintain parallel visual axes with and without glasses. Glasses then may be unnecessary. Other children appear to have weak accommodation. In them convergence excess is due to the extra stimulation required to increase accommodation to permit clear near vision. The use of bifocals is useful until the child's strength or that of the ciliary muscle is adequate.

A great many squints are only partially corrected when glasses are worn for long-sightedness. They appear to be a mixed type, the uncorrected portion of the squint being due probably to defective fusion (Figure III).

Type Associated with Defective Fusion.

The defective fusion group is the largest, but also the least understood, for the fusion sense and its centre, so certain to the last generation, are now looked on with suspicion. However, certain facts are known. (1) The inability to maintain binocular fixation in squint is usually associated

with an absence of the ability to fuse suitable objects that are presented to each macula. (ii) This ability to fuse can be produced by orthoptic training, or may develop automatically after an operation to straighten the eyes. (iii) The development of fusion by training may be associated with the appearance of the power to keep the eyes straight.

In the former types the paresis or the hypermetropia may be inherited. In this group it may be a greater strength of the internal than of the external rectus muscle that is transmitted. Frequently at operation the internal recti appear to be larger than their antagonists, the external recti muscles. This lack of binocular balance may break the immature bond of fusion, the excessive convergence taking charge before the fusion mechanism is sufficiently mature to hold its own.

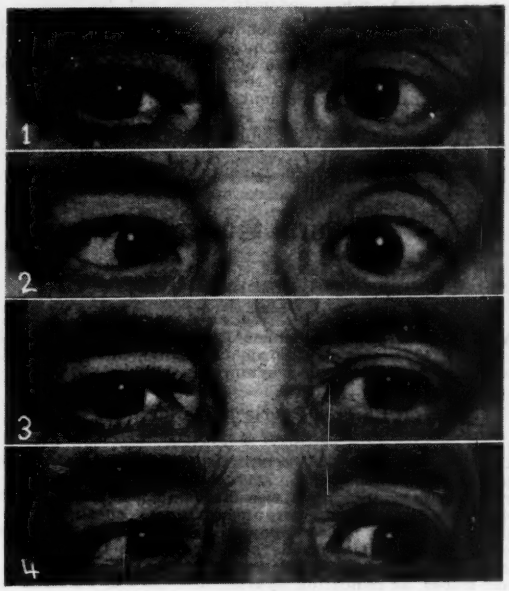

FIGURE II.

Left convergent squint which is due to paresis of the left external rectus. The angle increases while the subject looks to the left (2). Operation corrected the deformity (3 and 4).

The Treatment of Squint.

The objectives we seek are not only eyes that are parallel in all directions of gaze, but also the normal ability to fuse the images of objects seen by each eye into one picture. This alone permits the fullest degree of binocular vision and of the ability to judge distance and depth. This important mechanism of fusion may be obstructed by the following three defects. (i) Amblyopia or the inability to see clearly with the turned eye. (ii) Absent or anomalous correspondence between each retina. This means a failure of the object looked at to stimulate the corresponding cerebral cells in each side of the brain, appropriate stimulation of which permits fusion of the right and left ocular images and the building up of stereoscopic vision. (iii) Parallel or almost parallel visual axes. The most important of these defects is the first, for if it is not cured during the first few years of life the patient is vulnerable ever afterwards.

The First Obstacle: Amblyopia.

In a recent analysis the average age of onset of amblyopia was 1·7 years, and yet the average at the first visit to the oculist was 6·9 years. Here is a chance for some simple prevention of blindness! Of 75 children with convergent squints that appeared under the age of one year, 27 had amblyopia that persisted through life. It is impossible to estimate the number whose amblyopia was removed by early occlusion of the straight eye, for the ideal time to practise this is during the first two years of life—before the patient can give any indication of its vision. Because of this one relies on reversing the squint and keeping it alternating until the child is old enough for orthoptic training or surgery. As soon as a squint is diagnosed the refraction should be estimated, in case glasses will remove an excess of accommodation or improve vision. The eyes should be searched for an organic cause of visual defect. Then one should attempt to reverse the squint. It is often wise to use partial occlusion at first. One may commence with atropine drops instilled daily in the straight eye, and then one or more layers of "Durex" over the lids of that eye. If these measures fail, one proceeds gradually to complete occlusion with cardboard, "Elastoplast" or some other occluder. If the child lives remote from specialist treatment, this should be carried out temporarily by the practitioner concerned. Whilst the better eye is occluded, use of the other one should be encouraged with sewing cards, drawing, cutting-out *et cetera*.

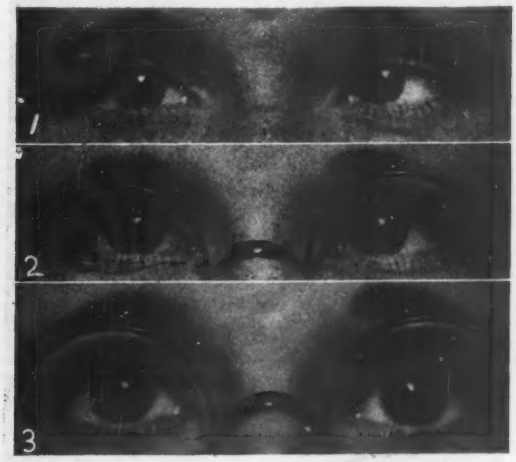

FIGURE III.

Partial accommodative squint (1). Operation completed the straightening (3) that was incomplete with glasses (2).

The "E" test can be used by parents for testing vision after three years of age. The letter is drawn on or cut out of cardboard and pinned up at the end of a room. As it is rotated, the child is asked to place its fingers or another cardboard "E" in the same direction as the test "E". In this way the vision of the two eyes may be compared.

The Second Obstacle: Absent or Abnormal Retinal Correspondence.

Of the remaining 48 children in the above-mentioned series of 75 with convergent squint arising under one year, 40 or over half (84%) had absent or abnormal correspondence. Fortunately this is a defect that can be eradicated later in life than can amblyopia.

The acquisition of binocular vision is a learning process—a mastering of conditioned reflexes. In man the phylogenetically older reflexes are present at birth, but those serving binocular vision are more recent and so are acquired in the first few years of life. If faults in the relevant structures or the child's environment are present, this acquisition may be slowed. If the delay is beyond a certain age, complete elaboration will not occur. By eight years the critical period is passing and the binocular reflexes may be said to be unconditionally fixed, so that after this age only gross anatomical lesions are capable of precipitating a deviation. Just as there is a great variation in the age at which people can learn foreign languages, so there is a variation, though more limited, in the age limits for acquiring retinal correspondence. The most important consideration in prognosis and treatment is the age of onset of the defect. Did it arise so early that the learning process had scarcely started? The next most important consideration is the age at which treatment starts. Is it early enough to bring to completion this arrested process before the learning period—the period of reflex flux—is over?

Operation can reproduce this period of reflex flux for retinal correspondence. It is interesting to find it becoming normal, even during the third decade, within a few weeks or months of operation.

The Third Obstacle: Parallel or Almost Parallel Visual Axes.

The third obstruction to the acquisition of binocular vision is the squint itself. For the majority of squints except those of small degree, of late onset, or of the intermittent or the accommodative types, surgery is necessary. The indications for and the purposes of surgery must be very carefully weighed when squint is variable or spastic in type or associated with amblyopia, or with under-accommodation or over-accommodation. The amblyopic eye may

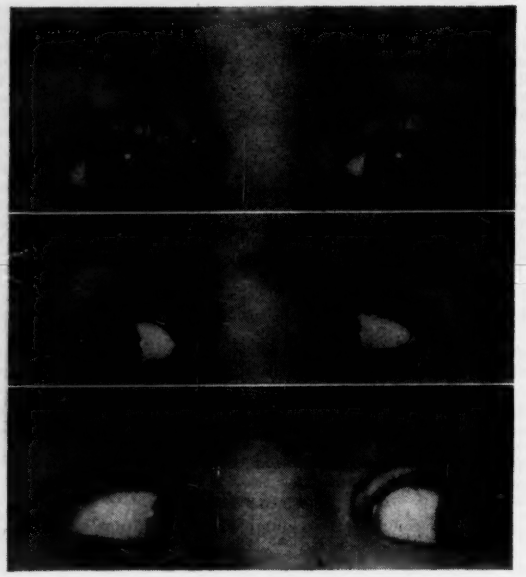

FIGURE IV.
Mongolian fold producing an apparent convergence which will disappear as the nasal bridge grows.

diverge; in later life a child may abandon glasses correcting long-sightedness, and the approach of presbyopia is inevitable. Accommodation then may fail to call on convergence, and the straight eyes of the teens may diverge. Parents should be told that frequently more than one operation may be desirable. Modern anæsthetics lessen the unpopularity of this remark. There are so many factors that cannot be estimated accurately that it is wise in many cases to plan for two operations. The strength of corresponding muscles of the two eyes varies, so does the strength of the same muscles in different individuals, and so, of course, does the size of the ocular globes. A five-millimetre recession of a strong muscle or on a small globe may produce a greater result than a similar operation on a weaker muscle or on a larger globe. In addition there is an occasional innervational tendency that leads to an unexpected post-operative deviation, usually vertical. However, this is less to be feared if vision is equal in both eyes and interretinal correspondence is normal.

Pseudo-Strabismus—A Warning.

A practitioner is sometimes disappointed and parents are surprised when the specialist disagrees with the former's diagnosis of squint. Not infrequently in the first few years of life there is an epicanthic or Mongolian fold in front of the inner canthus that gives the impression that the eyes converge, especially when the subject looks to either side (Figure IV). This disappears after a few years as the nasal bridge stretches the overlying tissue. More rarely eyes are set closer to the inner than to the outer canthi and the eyes appear to converge. If the practitioners would carry out a simple test, these conditions would be recognized and errors would not be made. The child should be seated opposite the doctor, who holds a torch before his own face to encourage fixation. Neither eye should move when they are covered alternately. The test is more accurate if the child will fix his gaze on a distant light. For example, if on covering the right eye the left moves outwards to fix the light, it must have been convergent before. If it moves inwards or downwards, it was divergent or upward before. (If the uncovered eye remains steady but the covered one moves outwards on being uncovered, a latent squint or esophoria is present.)

Conclusion.

A squinting parent is usually on the watch for a similar defect in the new baby. At two months the baby should begin to follow with both eyes a large moving object. The eyes should fix an interesting object at six months. During this period the "wind" of course can cause erratic movements. At the earliest age, however, any deviation that is constant must be taken seriously.

The specialist has still much to learn about squint. He and the patient are helped if he commences treatment early. He hears with alarm reports of children who have "grown out of" their squints, for in the lessening of the squinting there is probably a permanent lessening of sight in that eye.

J. RINGLAND ANDERSON,
Melbourne.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on July 9, 1952, at the Children's Hospital, Melbourne. The meeting took the form of a symposium on chronic pyuria.

Chronic Pyuria in Children.

Medical Aspects.

DR. H. NORMAN B. WETTENHALL, in presenting medical aspects of chronic pyuria, said that before the subject of chronic urinary infections was discussed, the term "pyuria" should be defined. In an uncentrifuged specimen six to eight white cells per low-power field, or in a centrifuged specimen two to three cells per high-power field, might be regarded as figures outside the upper limits of normal—in other words, as indicating pyuria. The clinical features were important, but the changes found microscopically in the urine were fundamental. The condition of pyuria was probably more common than was generally realized. According to Mitchell and Nelson it was responsible for 1% to 2% of all admissions to children's hospitals. In the years from 1945 to 1951, 45 to 50 children had been admitted each year with that condition to the Children's Hospital, Melbourne. Those figures did not include children admitted to hospital with congenital anomalies of the renal tract in whom no infection was present at the time of their admission; nor did they include the many patients attending out-patient clinics who were not admitted to hospital. Six times as many girls as boys over the age of two years were affected. Under that age the ratio between girls and boys was two to one. Discussing the ætiology, Dr. Wettenhall said that bacteriologically there were a variety of organisms which could cause infection of the renal tract. Bacilli of the colon group, including *Bacillus aerogenes*, were the causative organisms in 80% of cases. Streptococci and staphylococci were the infecting organisms in 15%, and miscellaneous organisms such as *Bacillus proteus* and *Pseudomonas pyocyanea* made up the remaining 5%. Infections in which mixed flora were grown on culture were commonly associated with urinary stasis. In conditions in which there was persistent pyuria and no organism was grown on culture, tuberculosis should always be suspected. The infection could be blood-borne, but was frequently an ascending infection from the lower part of the urinary tract, and that was why the condition occurred more commonly in girls than in boys.

Dr. Wettenhall went on to say that understanding of the basic pathology was necessary for correct appreciation of the management of those children. The term "pyelitis" commonly used for patients in whom infection of the urine was recognized, was likely to be misleading. The infective process was not confined to the pelvis, but extended throughout the interstitial tissues of the kidney. Microscopic examination revealed collections of inflammatory cells

including a high proportion of polymorphonuclear leucocytes. Those were spread diffusely or focally, and in the more severe infections were sometimes so extensive that they showed macroscopically as small abscesses. It was evident that the term "pyelonephritis" was preferable to "pyelitis". The course of the infection could be that of any inflammation—namely, resolution, healing by fibrosis, or destruction of areas of kidney tissue. When the infection continued or recurred over many years, destruction and distortion of renal tissue were often so pronounced that the picture was difficult to distinguish from the small contracted kidney of chronic glomerulonephritis. In some cases pyuria was due to infection of the urethra and bladder, but clinically this was often impossible to distinguish from low-grade pyelonephritis.

Referring to the symptomatology, Dr. Wettenhall said that the acute onset with high fever, rigors, lumbar pain and frequency and scalding on micturition was the classical picture presented by a patient with urinary tract infection. However, there were more insidious types of onset which applied particularly to infants. Failure to thrive and vomiting were not uncommonly the presenting symptoms in infancy. All babies with persistent vomiting of doubtful ætiology should have urine examinations. At times urinary infections could complicate other illnesses such as meningitis or gastro-enteritis. In older children worms were often associated with persistent pyuria. The old adage of "more is missed by not looking than by not knowing" should never be forgotten in the examination of children suspected of having a urinary tract infection.

Dr. Wettenhall then said that urinary tract infections were notorious for the number of times they recurred, and in a number of cases low-grade infections between apparent recurrences could persist unrecognized by the medical practitioner. The persisting or recurring infections resulted in further renal damage, so that ultimately the symptoms and signs of renal failure developed. That stage was often long delayed, and many children grew up to live a normal adult life. However, it was impossible to be dogmatic about what was likely to happen to a particular patient, and all affected children should be regarded with the same seriousness as far as treatment was concerned.

Discussing treatment, Dr. Wettenhall said that the goal of treatment was eradication of the infection rather than simple sterilization of the urine. Investigations to determine the causative organism should always be performed, and direct culture of the urine should precede the institution of chemotherapy. Once the organism was recovered it should be tested for sensitivity to antibiotics. In the first attack of pyelonephritis the particular antibiotic used was of less importance, but when an infection persisted or recurred the correct antibiotic was essential, otherwise it was not uncommon for the overgrowth of a resistant organism to occur. Antibiotic therapy should be continued for a least a week in an initial attack, or for up to three weeks in any recurrence. Adjuvant measures were the giving of free fluids and possibly the alteration of urinary pH to inhibit bacterial growth. That method, however, had fallen into some disrepute, since for it to be effective it was necessary to establish a pH of 9·2 on the alkaline side, a level which could not be obtained with the dosage of potassium citrate usually ordered; on the acid side a pH of 5·2, which could be achieved with mandelic acid therapy, was required. General hygiene was most important. The taking of a bath at least once a day was an obvious necessity for cleanliness, but was a habit not yet adopted by all families. The underwear of little girls often left much to be desired, and it was not uncommon to find that their undergarments were screwed up and wedged in the vulval opening. Another cause of vulval irritation was infestation with threadworms. In such cases institution of appropriate therapy would often clear up the pyuria. Elimination of foci of infection such as infected teeth and tonsils had been a very popular method of therapy in the past, and although nowadays often held in disrepute, still occasionally brought cures. Investigations in all cases of chronic pyuria should be carried out to determine the state of renal function. Such measures included a urea concentration test and water excretion tests, in addition to ordinary ward testing of urine. Anatomical abnormalities were not uncommonly associated with chronic pyuria, and investigation for these was most important.

Surgical Aspects.

DR. F. DOUGLAS STEPHENS, in discussing surgical aspects of chronic pyuria in children, said that the purpose of the paper was to discuss the surgical cases of chronic pyuria investigated in the genito-urinary clinic during the past two years. Two special X-ray techniques in frequent use were to be described and their special value demonstrated. Thirty-eight cases had been collected and the material had been classified and presented. The routine surgical urinary investigation had been carried out in all cases of chronic pyuria. That included examination and culture of the urine, cystoscopy, urethroscopy, retrograde pyelography and excretion pyelography. In addition, the following two special urological investigations were frequently used—"Wydase" (hyaluronidase) pyelography and micturition urethrography. "Wydase" pyelography had replaced intravenous pyelography in the examination of infants up to two years of age, whose veins were so small that the insertion of needles was difficult. The spreading factor hyaluronidase enhanced the absorption of iodide from the tissues. Three hundred turbidity reducing units of "Wydase" (Wyeth) were dissolved in two millilitres of 2% procaine solution and 18 millilitres of 35% "Uriodone" solution. For an infant aged six months 14 millilitres, and for a child aged twenty-four months 20 millilitres, were used. A preliminary cutaneous iodine sensitivity test was carried out twenty-four hours prior to the investigation. Routine films were taken at intervals until the greater proportion of the iodine had been absorbed in approximately fifty minutes. Dr. Stephens said that, using that technique, he had been satisfied with most of the results. Describing micturition urethrography, he said that after the urine had been withdrawn 16% "Uriodone" solution was injected into the bladder through a small catheter. Sufficient was introduced to cause an urgent desire to micturate. The catheter was then removed and skiagrams were taken of the urinary tract prior to and during micturition. The method had advantages over the retrograde urethrography, in which the posterior part of the urethra was partially occluded owing to reflex spasm of the *compressor urethræ* during the injection. That method demonstrated the physiological distension of the posterior and penile parts of the urethra during micturition, the contours of the bladder at rest and during the effort of micturition, reflux of bladder contents into diverticula or into dilated ureters, and in immediate and delayed post-micturition films the distinction between true and false residual bladder contents. True residual urine was that which remained in the bladder immediately after a completed voluntary act of micturition. False residual or reflux residual urine was that which was expelled shortly after micturition back into the bladder from dilated ureters or diverticula, into which it had entered by reflux during micturition.

Discussing surgical causes of chronic pyuria, Dr. Stephens said that the two primary factors were prolonged retention of urine in the urinary tract due to incomplete evacuation, and chronic parenchymal abscesses. The chief causes of retention of urine were obstruction, reflux residual urine, and detrusor paralysis of the bladder. In a series of 14 boys obstruction of the urethra was the cause of pyuria. The obstruction was due to valves of the urethra in six cases. The valves occurred at the lower end of the verumontanum. The free edges of the valves were the limbs of the bifurcated inferior *crista urethralis*, which diverged in an oblique direction around the lateral walls of the urethra. In three of those the valves were successfully resected with the infant McCarthy resectoscope. In two cases a urethral membrane occurred in the region of the triangular ligament at the distal end of the membranous urethra. Both the patients were newborn infants who died shortly after birth. The frailness of the membrane suggested that successful treatment was probable by dilatation only. A large mucous cyst in the base of the bladder partially obstructing the urethral outlet was the cause of stagnation of urine in the bladder in one case. The cyst was excised through a suprapubic incision. There were two cases of diverticula of the penile portion of the urethra causing partial urethral obstruction. The diverticula enlarged considerably during micturition, and in two cases the diverticula were shown to have occluded the urethral lumina. Two other patients with urethral obstruction had had many forms of treatment, and investigation had failed to reveal the original cause of the obstruction. Dr. Stephens went on to say that obstructions at the upper end of the ureter and at the pelvi-ureteric junction were well-known causes of hydronephrosis and of pyuria and for that reason would not be discussed. Unilateral stricture at the vesico-ureteral junction had occurred in two girls and one boy. The strictures were demonstrated by intravenous pyelography and by ascending pyelography. All patients underwent operations on the lower ureteric orifice, and the condition of two was now satisfactory. One required removal of the kidney and the ureter subsequently, owing to recurrent infection due to vesico-ureteric reflux. Three cases of ureterocele had occurred; in one the condition was bilateral, in one unilateral and in one on an accessory ureter. It was shown on cysto-urethrographic examination that during micturition the intravesical cystic dilatation became flattened out and even

evaginated from the bladder and was not causing obstruction to the urethral outlet. Dilatation of the uninvolved ureters occurred in the unilateral cases, and it was concluded that the orifices must have been partially occluded by the distended intravesical cystic swelling of the opposite ureteroceles. One patient with bilateral ureterocele died; the remaining two were treated successfully, one by incision of the ureterocele, and the other by incision of the ureterocele together with excision of the accessory ureter in the other.

Reflex residual urine—defined as the urine which flowed back from the bladder into the ureters or into a diverticulum during micturition, and which was then expelled back into the bladder after micturition—differed from true residual urine, which was the urine remaining in the bladder after an attempt at complete evacuation, as in urethral obstruction and vesical detrusor weakness. Dr. Stephens said that bilateral idiopathic hydroureter had occurred in five females. There was free reflux into the ureters and free expulsion from the ureters into the bladder. There were two cases in which operation on the ureteric stricture was followed by reflux. The treatment of this condition was non-operative. It was apparent from the micturition urethrography that the bladder function was normal, but the valve-like action of the lower ends of the ureters was deficient, permitting reflux. The bladder and ureters could be almost completely evacuated by three successive attempts at micturition at half-minute intervals. If the child was old enough to cooperate, that form of treatment was probably satisfactory. Reflux occurred into diverticula of the bladder. There were three such cases, two associated with urethral obstruction and one without. One patient died, one had the urethral obstruction and the diverticulum successfully removed, and one was awaiting operation. Distension of a diverticulum in close proximity to the lower end of the ureter caused partial obstruction and hydroureter by compression of the ureteric lumen. That added to the total residual urine. Detrusor paralysis was found in paralysis of the *nervi erigentes* at pelvic operations, in obvious lesions of the spine such as spinal meningomyelocele, and in one case of polyneuritis following pink disease. Into that group was placed temporarily one case of retention of urine in which no bladder neck or urethral abnormality was found on micturition cystourethrography, and in which the condition was called detrusor incoordination of the bladder in the absence of any other structural anomaly. Treatment of the condition was directed towards assisting evacuation by manual pressure in the suprapubic region.

Referring to congenital anomalies, Dr. Stephens said that pyuria occurred in five cases of double ureter, and the reason for the infection was not well understood in four in which the ureters were normal in calibre. In two cases the ureter to the upper atavistic segment was ligated and divided, and reinfection occurred in one. In two cases the upper segment of the kidney together with the greater portion of the ureters had been removed, and infection recurred in one. In one case no operation had been performed.

One case of chronic abscess of the kidney due to tuberculosis, in which a local abscess was demonstrated radiologically and was considered too large to treat with streptomycin alone, occurred in the series; nephrectomy had been performed with satisfactory results. A more conservative trend was apparent in the literature, and a good case had been made for segmental resection of the abscess under streptomycin cover.

Discussion.

DR. ROBERT SOUTHBY, in opening the discussion, pointed out that it was important to determine that pyuria was a result of urinary tract infection, and not due to a condition such as a foreign body in the vagina in small girls or balanitis in small boys. In his experience, true rigors in small children were usually due to pyelonephritis. Permanent renal damage with destruction of parenchyma and scarring was often the sequel of chronic urinary tract infection. Owing to the large functional reserve capacity of the kidneys, symptoms were frequently few until just before the terminal uræmic stage.

Dr. Southby confirmed Dr. Wettenhall's statement that failure to thrive and vomiting in small babies were often the only features of chronic urinary tract infection. Recurrent febrile episodes, even though mild, with few pus cells found on urine examination, should be vigorously treated, if the same organism was grown on culture on several occasions. Treatment should be specific as determined by sensitivity tests. Failure to use such tests could lead to the development of strains of organisms resistant to many antibiotics, and was incidentally uneconomical. Dr. Southby said that persistent pyuria in newborn babies was suggestive of congenital abnormalities. Tuberculous infection of the kidneys must always be considered in the diagnosis of pyuria of obscure origin. The presence of poor urinary concentration and a urinary specific gravity fixed within the range of 1·004 to 1·008 indicated permanent renal damage. Dr. Southby quoted cases of urinary tract infection in the same family, and asked Dr. Wettenhall if he had any experience of familial incidence.

Dr. Wettenhall, in reply, said that in one instance only had he discovered two cases of urinary tract infection in one family.

DR. HAROLD MOORE stressed the need for specific therapy as determined by sensitivity tests. He said that sometimes the original pathogenic organism disappeared, but was replaced by another, such as *Bacillus proteus*. Tuberculosis of the kidney was not a common disease in childhood, and usually did not appear until late in the second decade. Once a tuberculous abscess was radiologically demonstrable, it would not respond to anything but surgical removal. That usually meant nephrectomy, but recently surgical removal of the abscess itself had been performed. Urinary tract infection associated with a double ureter was usually a result of infection of the upper ureter, which frequently opened in abnormal situations from which ascending infection was simplified.

DR. L. P. WAIT said that some cases of urinary tract infection in childhood were due to renal calculi, and quoted two cases, in one of which almost complete destruction of both kidneys was demonstrated at autopsy.

DR. V. L. COLLINS pointed out that the term "chronic pyuria" suggested that the condition was easily recognizable. However, on occasions no pus was detectable microscopically, and the diagnosis could be made only on culture of the urine. The correct diagnosis was important, as renal failure could insidiously develop without apparent cause. Pyelitis probably did not occur without renal involvement. Dr. Collins asked Dr. Wettenhall why there was frequently an absence of hypertension in patients in whom, at autopsy, grossly scarred kidneys were found.

Dr. Wettenhall replied that he did not know the answer.

DR. MEDWYN HUTSON asked Dr. Stephens if there was any treatment for idiopathic hydroureter with residual reflux.

Dr. Stephens replied that surgical measures would not improve the condition.

DR. DOUGLAS B. DUFFY said that in his experience very few adults with chronic urinary tract infections presented a past history of infection in childhood, and asked if there were records of follow-up examination of children with that condition.

Dr. Wettenhall replied that in a series of approximately 20 patients who were followed up, there was a high proportion who had either persistent urinary tract infection or renal damage in adult life. Other reported series confirmed that observation.

Dr. Duffy remarked that the cause of hydroureter with residual reflux was unknown, and the treatment was medical. It was well to remember that the results of in-vitro sensitivity tests to antibiotics often failed to correspond to the in-vivo effects. A combination of antibiotics was frequently more successful than one used alone.

DR. KEITH HALLAM pointed out that not only did calculi cause the development of renal tract infection, but the converse also occurred. It would be interesting to trace the childhood history of adults with renal calculi. With reference to the condition of double ureter, Dr. Hallam said that if the upper ureter opened into the lower ureter at a site where peristaltic contractions occurred, intermittent obstruction could develop. That could explain the fact that the upper ureter and the upper pole of the kidney were the ones usually affected.

DR. JOHN PERRY said that he considered the term pyelitis referred to a clinical rather than a pathological condition. Although the results of in-vitro sensitivity tests did not correspond to in-vivo effects, sensitivity tests should be carried out before indiscriminate therapy was commenced.

DR. STANLEY WILLIAMS asked whether sulphonamide therapy was contraindicated in pyelonephritis.

Dr. Wettenhall, in reply, said that the infection involved the interstitial tissues of the kidneys, and that the plasma levels of antibiotics rather than their urinary concentrations were important. Sulphonamide therapy was contraindicated only if renal function was impaired, and those sulphonamides which gave the highest plasma concentra-

tion on standard doses were preferable. The course of treatment in chronic urinary tract infections should be two to three weeks in duration.

Dr. Williams inquired about the management of patients with double ureter.

Dr. Stephens, in reply, said that if the condition was symptomless and there was no pyuria, no treatment was necessary. However, if pain or pyuria from the affected side developed, surgical treatment was indicated—removal of either part or whole of the affected kidney.

DR. A. P. DERHAM stressed the frequency of urinary tract infections in small children, and agreed that pyelonephritis was the only common condition in childhood to present itself with a true rigor.

DR. HENRY SINN asked Dr. Wettenhall for his opinion on the value of small doses of sulphonamides or antibiotics given over a long period for preventing recurrence or an acute exacerbation of a chronic urinary tract infection, especially in those cases in which surgical treatment was contraindicated.

Dr. Wettenhall, in reply, said that he was unable to give a definite answer, but he himself had used sulphonamides for that purpose and was of the impression that they were of use in preventing recurrences. At least in his experience no harmful effects had been noticed.

DR. JOHN COLEBATCH asked Dr. Stephens the means by which tuberculous renal infection of minimal severity could be diagnosed.

Dr. Stephens said that the diagnosis depended on the growth of a culture of *Mycobacterium tuberculosis* from the urine.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

ACTING GOVERNOR SNODGRASS TO LORD GLENELG.[1]

[*Historical Records of Australia.*]

Government House, Sydney,
22 Feby., 1838.

My Lord,

It is my painful duty to inform Your Lordship that the ship "Minerva", which arrived in this Port upon the 23rd Ultimo with Emigrants sent out by private Individuals with Bounties, was found to be affected by a contagious fever, rendering it necessary to place the vessel and Emigrants under Quarantine. The vessel with a portion of the Emigrants was released on the 12th Instant but the remainder of the Passengers are still under restraint at the Station at Spring Cove.

The accompanying Copies of the Report of the Medical Board upon the state of the vessel on arrival and a further Medical Report received of the latest date, together with a list of the deaths which have taken place during the voyage and subsequently will place Your Lordship in possession of early information upon the melancholy subject.

I have, &c.,
K. SNODGRASS.

Report by Medical Board.

Deputy Inspector General's Office,
25th January, 1838.

The Medical Board on the Emigrants per "Minerva" having assembled and taken into consideration the report of 24th instant from Assistant Surgeon Stuart in charge, have to express their regret that the real State of the Emigrants from the documents (herewith) is much more unfavorable than anticipated from the previous Statement of the Captain.

By this report it appears that the disease first manifested itself about the 25th November, since which eighty six have been attacked: of these fourteen have died and there now remains sixty independent Convalescents, thirty four of which a list appended.

The Board consequently would recommend that the whole of the Emigrants should be landed as speedily as circumstances will permit, the Sick and Convalescents apart from the rest, and placed under the usual restrictions and regulations. And that the tween decks of the ship shall be cleared and thoroughly cleansed and fumigated (etc) taking care that none of the fittings up be employed in the Houses or Tents ashore, at least until they have been well scrubbed with Sand and Soap under the immediate Superintendence of the Surgeon in charge.

The Board are further of Opinion that it would be desirable to place all the families, in which the disease has hitherto occurred, together in one or two of the Houses, so as to keep them in Some measure apart from the rest, by which means the further Spread of the disease is likely to be more Speedily checked.

J. V. THOMPSON,
Deputy Inspr General Prest.
J. H. DU MOULIN,
Surgeon 50th Regt and forces.
K. ROBERTSON,
Col Surgeon.

A further report from the Board dated 24th February 1838 is as follows:

"Have to observe that no death has occurred, nor any new case, since the previous report, and that the Sick in Hospital are rapidly diminishing and going on favourably."

However, the enclosure shows 24 deaths prior to February 22, 1838.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

LOCAL USE OF ANTIBIOTICS IN OPHTHALMOLOGY.

SIR: It is impossible, to coin yet another philolologically unattractive word, to "aseptisize" the conjunctival sac. But no apology is made, for disease is unattractive, and to fight it we must sometimes use unattractive methods; to describe our efforts to combat its protean manifestations, we are often forced to employ synthetic and polysyllabic words and phrases, which are discordant to the purist ear.

Within the folds and fornices of the bulbar and palpebral conjunctival mucous membrane, there dwells a myriad host of unicellular and multicellular organisms, together with those mysterious elements that we comfort our lack of omniscience with, by calling ultra-microscopic viruses.

Anyway, there is a whale of a lot of motile organisms around; some float down from the lachrymal gland, some surge up from the nasal meatuses via the lachrymal sac, and some just drop in from the wind that bloweth where it listeth. Some are saphrophytic, some are pathogenic, and others, like the "floating vote", are time servers, base opportunists, who wait to see which way the wind of health blows. From all of these, many are called, by Nature's still mysterious processes, but only a few (millions) are chosen.

Among the few chosen, there are sometimes present dour enemies of the common ocular weal, and all the arts and crafts of the skilled pathologist may fail quantitatively to enumerate them. For what shall it avail, if smear and culture find a preponderance of the innocuous *Staphylococcus albus*, when a small but vigorous minority of *Staphylococcus aureus*, or *Streptococcus hæmolyticus*, or filter-passing viruses, lie hidden in the undergrowth beneath the luxuriant foliage of the innocent flora?

And what shall it avail the doctor or, more importantly, the patient if, during the progress of an intraocular operation, ten million harmless organisms stream into the aqueous humour and only ten thousand harmful elements slide in too? What is the doctor to do when he sees the swollen eyelids, the hazy cornea, the turbid aqueous, the absent or misty vision and the distress of his patient? It is a bit late then for sulphonamides, antibiotics, heat lavage *et cetera*. Only prayer remains, sometimes mercifully answered; sometimes, alas, unanswered.

Is it not better to use an antibiotic solution before, during and after operation? In my hands and in those of many other oculists, the routine pre-operative local use of an adequate antibiotic solution has reduced the incidence of post-operative infection by at least 75%.

I agree wholeheartedly with Dr. V. M. Coppleson (THE MEDICAL JOURNAL OF AUSTRALIA, June 28, 1952), who said that the strength of the antibiotic brought into contact with the organism was an important consideration in the question of the development of resistance. In the same issue of the journal, my distinguished colleagues Dr. Hales Wilson and Dr. Edgar Thomson point out some of the dangers of antibiotics; with their opinions and warnings, all must agree. They have told the truth and nothing but the truth; have they told the whole truth? Have they, with all sincere respect for their warnings, pointed out the dangers, in certain circumstances, of not using antibiotics?

Yours, etc.,

135 Macquarie Street, Sydney, ARTHUR D'OMBRAIN.

August 5, 1952.

RETIREMENT OF PROFESSOR W. K. INGLIS AND PROFESSOR H. K. WARD.

SIR: It may not be known to all graduates of the University of Sydney medical school that the professors of pathology and of bacteriology, W. Keith Inglis and Hugh K. Ward, have recently retired from their chairs after long and valuable service. Many of their contemporaries will remember them in their student days. Both got a blue for rowing, and Ward went on as Rhodes Scholar to Oxford and stroked the Australian Olympic crew at Stockholm. Both were on active service in France in the first World War. Inglis gave many years to the Sydney Hospital as pathologist and to the university as demonstrator and lecturer in pathology before he attained the professorship, whilst Ward spent many years at Oxford and at Harvard before he was appointed professor of bacteriology. So both had a very sound training and background and had published much when they took up their appointments, and many generations of students, now graduates and in practice, will remember the teaching they gave and how earnestly and loyally they stuck to their job. Both were also elected members of the Council of the British Medical Association, and Inglis was President in his time.

Some months ago at a meeting of the Section of Pathology a committee was formed to take steps to mark their retirement in a manner worthy of their contributions to the subjects of pathology and bacteriology in Australia and their services to the medical school and profession in general. The appeal has been so far almost limited to pathologists, but some of our colleagues think that we should make a wider appeal to many members of the profession who are friends and admirers of the retiring professors. Our committee was very glad to receive and accept this suggestion.

Contributions should be sent to the treasurer, Dr. V. J. McGovern, Fairfax Institute of Pathology, Royal Prince Alfred Hospital, who will acknowledge them, and contributors will later be invited to the presentation.

Yours, etc.,

143 Macquarie Street, Sydney, A. H. TEBBUTT.

October 22, 1952.

THE COMPLICATIONS OF ORO-TRACHEAL ANÆSTHESIA.

SIR: Associated with the frequent administration of muscle relaxant drugs, the use of oro-tracheal tubes in anæsthesia has increased and offers many advantages. Besides thoracic surgery and most surgery of the head and neck, there are many other indications for the use of oro-tracheal anæsthesia. However, it is the complications of this technique that I wish to emphasize and so stress the importance of anæsthetic training if the number of preventible anæsthetic deaths is to be reduced.

The complications of oro-tracheal anæsthesia may be divided into three periods: (i) intubation; (ii) the progress of the operation; (iii) extubation and post-operatively.

Complications of intubation are: (i) Trauma to (*a*) lips, causing ulceration; (*b*) teeth, causing a foreign body danger; (*c*) pharynx, which may result in surgical emphysema; (*d*) vocal cords, which may result in ulceration; (*e*) trachea, which may result in tracheitis and œdema. (ii) Laryngeal or tracheo-bronchial spasm. (iii) Initiation of abnormal cardio-tracheal reflexes causing cardiac arrhythmias. (iv) Tube in œsophagus may result in rupture of the œsophagus or stomach.

Complications during operation are: (i) Kinking of tube, especially on posturing patient. (ii) Respiratory obstruction due to too small a tube. (iii) Slipping out of tube from trachea due to inadequate fixation. (iv) Intubation of right bronchus due to too long a tube. (v) Herniation of defective cuff over end of tube causing obstruction. (vi) Pressure of cuff on soft lumen of tube causing obstruction.

Complications on extubation and post-operatively are: (i) Laryngeal spasm. (ii) Laryngeal œdema, which may result from trauma of intubation, too large a tube for too long a time, infection or sterilizing irritant on the tube. (iii) Granuloma of cord.

A technique associated with so many complications should not be used unless definitely indicated and not by the inexperienced anæsthetist except in an emergency.

Dr. Ralph Waters summed up his experiences as follows: "The incidence of deaths and major complications appears to be due, not to the nature of the drug chosen or the method of its administration, but to lack of training and inexperience on the part of the anæsthetist."

Yours, etc.,

G. V. STANTON, M.B., B.S., D.A.,
Honorary Anæsthetist, Royal Melbourne Hospital.

Parkville, N.2, Victoria,

October 23, 1952.

ULTRASONIC ENERGY IN PHYSICAL MEDICINE.

SIR: With reference to your leading article "Ultrasonic Energy in Physical Medicine" in the journal of August 23, 1952, I wish to draw your attention to the fact that it is not usual in scientific circles to condemn a method of treatment on the findings of a single group of investigators.

It would be interesting to know whether the writer of the leader has acquainted himself with the vast European literature or has any practical experience with ultrasonic treatment, so that he is really able to come to the conclusion that "a good deal of the European work seems to be uncritical".

The same experiments as those carried out in Minnesota have been done prior to 1949 by Peters, Schmitz, Horvath, Lennert and many others (*"Der Ultraschall in der Medizin"*, Hirzel, Zurich), and nothing new is revealed by the findings of the Mayo group. As Pohlman answered similar critics so aptly in his *"Die Ultraschalltherapie"* (Huber, Bern, 1951), I will translate his words:

> Experiments on animals carried out by Buchtala, Barth, Bülow, and Waschmann on puppies, rats, mice, small fishes and tadpoles show that even low intensities may cause damage and even death. This does not apply to man, as . . . the intensities used represent an incomparably high dosage for these small animals. Nobody would imagine that he could cause damage to an elephant with such intensities. If we do not allow the comparison for larger animals we cannot use it for smaller ones. The size ratios elephant : man and man : puppy are about the same. Adam treated over 1500 children and never had a single case of bone damage in any form. . . .
>
> We must not forget that if overdoses are employed, any form of energy will cause damage. If this were not the case, the energy would be biologically ineffective and thus useless. Doctors would hardly advise abolishing X-ray therapy, because it is possible to burn a patient with an overdose. It is almost unbelievable that this argument should be used against ultrasonics.

In 1938 Pohlman began a series of experiments. He and his assistants applied the ultrasonic head to their own bodies and raised the intensity until it caused damage. They found that the dose necessary to produce blistering or necrosis caused almost unbearable pain during treatment. They were thus able to make a scale of therapeutic dosages well below the pain and danger level. With four watts per square centimetre they treated the palms of their hands, where the bones are comparatively superficial, with a slightly moving head for a period of ten minutes daily for six months, in order to see whether there was any ill effect from constant and repeated treatment over a long period.

After ten years, in spite of repeating this six months' treatment, there have been no ill effects at all. Pohlman found that a dosage of five watts per square centimetre with a moving head was within the safety margin.

I have personally worked in one of the foremost European clinics and have seen how conscientiously the results were classified. What the Mayo authors call "enthusiastic European reports" can be well supported by evidence, and even in my own small country practice where I am applying ultrasonic treatment with uniformly good results, especially in the rheumatic group of illnesses, this can be substantiated.

One of the reasons why sometimes unsatisfactory results are reported is that some of the commercial machines on the market do not produce a high enough intensity to have any effect except in the most superficial lesions, or do not generate modulated current, or have other disadvantages. A further reason may be that the operator is not fully acquainted with the technique. As regards the remarks on tumours in the report, ultrasonics has its contraindications like any other form of therapy and tumours are one of them.

In 1949 there was already a recorded number of over 9500 cases treated in 29 clinics, including university clinics in France, Switzerland, Germany and Austria, which had been treated without any damage. Since then the number of cases treated runs into tens of thousands. It seems rather late in the day for the American investigators to call for an investigation when it has been already done years ago, and come to the sweeping conclusion that "not enough is known concerning the physiological effects of ultrasonic energy to warrant its use in any disease of man".

I am rather surprised that THE MEDICAL JOURNAL OF AUSTRALIA should give publicity apt to retard the progress of a valuable therapeutic agent without taking the trouble to examine the evidence on both sides.

Yours, etc.,

ELLA CHAMBERS.

Laurieton,
New South Wales,
October 20, 1952.

Notice.

ALFRED HOSPITAL RESIDENTS AND GRADUATES' ASSOCIATION.

THE Alfred Hospital Old Residents' Association has recently changed its constitution so that all students who graduated from the clinical school of the Alfred Hospital may become members of this Association, whose name is now as shown above. Any ex-student of the Alfred Hospital, Melbourne, is invited to contact either of the joint honorary secretaries, Dr. G. Raleigh Weigall and Dr. R. S. Smibert, for further information about membership.

The Royal Australasian College of Physicians.

New Members.

AN examination for membership of The Royal Australasian College of Physicians was held in Australia in October, 1952, and the following successful candidates were admitted by Council to membership of the College: Dr. John Beveridge, Dr. R. S. Colton, Dr. M. Faunce, Dr. A. K. Garven, Dr. D. F. King, Dr. G. S. Nagy, Dr. J. Palmer and Dr. I. S. Wallman.

Examination for Membership.

An examination for membership of The Royal Australasian College of Physicians will take place in April-May, 1953. The written examination will be held on Saturday, April 18, in capital cities of the Commonwealth where candidates are offering. The clinical examination will take place in Melbourne on approximately Thursday and Friday, May 28 and 29, 1953.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 11, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.[2]	Northern Territory.	Australian Capital Territory.	Australia.[3]
Acute Rheumatism	2(1)	..	..	..	..	..	..	..	2
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	9(9)	..	..	..	..	..	..	9
Diphtheria	3(1)	4(2)	4(2)	..	2(2)	..	..	..	13
Dysentery (Bacillary)	..	..	2(2)	..	..	..	..	..	2
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	..	..	..	7(6)	..	..	..	7
Lead Poisoning	..	..	..	..	1	..	..	..	1
Leprosy	..	2(2)	1	..	5	..	..	..	8
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	3(3)	7(5)	5(1)	..	..	..	..	..	15
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	1(1)	..	..	..	..	..	..	1
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	4(3)	10(1)	..	7(5)	1	..	..	..	22
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	40(31)	..	..	1	..	..	3	44
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	12(6)	20(16)	9(5)	2	4(3)	..	..	..	47
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	25(18)	18(14)	10(7)	8(8)	6(2)	..	..	..	67
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	1(1)	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Tasmania.

Only those candidates whose answers in the written examination have attained a satisfactory standard will be asked by the Censor-in-Chief to proceed to the clinical examination.

Applications to appear before the Board of Censors should be made in the prescribed form and must be in the hands of the Honorary Secretary of the College not later than Saturday, March 21, 1953. Application forms are obtainable from the Honorary Secretary, 145 Macquarie Street, Sydney.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, promotions *et cetera* are promulgated in the *Commonwealth of Australia Gazette*, Number 68, of October 2, 1952.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Promotion.—Acting Interim Surgeon Commander Robert Enwright Lauder is promoted to the rank of Surgeon Commander, dated 30th June, 1952.

Resignation.—The resignation of Joseph Lee Watson of his appointment as Surgeon Lieutenant-Commander (for short service) is accepted, dated 31st October, 1952.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Volunteer Reserve.

Transfer to Retired List.—Surgeon Commander Reginald Francis Matters is transferred to the Retired List, dated 22nd July, 1952.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The probationary appointment of Pilot Officer D. B. McKillop (024158) is confirmed, and he is promoted to the rank of Flying Officer, 30th April, 1952.

The surname of Flight Lieutenant Heino Borkan (025627) as approved in Executive Council Minute No. 60 of 1952, appearing in *Gazette* No. 55, dated 14th August, 1952, is amended to read "Borkman".

Obituary.

SIR CONSTANTINE TRENT CHAMPION DE CRESPIGNY.

WE regret to announce the death of Sir Constantine Trent Champion de Crespigny, which occurred on October 27, 1952, at Adelaide.

Corrigendum.

IN THE MEDICAL JOURNAL OF AUSTRALIA of October 18, 1952, at page 554, an error appears in the summary of the president's address delivered by Dr. John Hoets to the Section of Orthopædics and Physical Medicine of the Australasian Medical Congress (British Medical Association), Eighth Session. The words "Septic necrosis" in the second line of the second column of the summary should read "Aseptic necrosis". We regret this error.

Medical Appointments.

Dr. J. Davis has been appointed a member of the Board of Optometrical Registration of New South Wales.

Dr. R. R. Webb has been appointed psychiatrist in the Mental Hygiene Branch, Department of Health, Victoria.

Dr. R. D. Wilson has been appointed public vaccinator for the City of Prahran, Victoria.

Dr. J. H. Steel has been appointed government medical officer at Winton, Queensland.

Dr. A. R. Doutreband has been appointed to the State Hospitals and Homes Branch, Department of Public Health, New South Wales.

Dr. E. S. A. Meyers has been appointed to the staff of the Department of Public Health, New South Wales.

Diary for the Month.

Nov. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 17.—Victorian Branch, B.M.A.: Finance Subcommittee.
Nov. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 19.—Western Australian Branch, B.M.A.: General Meeting.
Nov. 20.—Victorian Branch, B.M.A.: Executive Committee.
Nov. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 25.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 26.—Victorian Branch, B.M.A.: Council Meeting.
Nov. 27.—New South Wales Branch, B.M.A.: Branch Meeting.
Nov. 27.—South Australian Branch, B.M.A.: Scientific Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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